JAN

Access DB# 86845

SEARCH REQUEST FORM

Scientific and Technical Information Center

Requester's Full Name: FONDA Examiner #: 71970 Date: 2-14-03
Art Unit: 1623 Phone Number 30 8-1620 Serial Number: 09/937110
Mail Box and Bldg/Room Location: 8B19 8A05 Results Format Preferred (circle): PAPER DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need.

****

Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: see attached sheets — no assignment

Inventors (please provide full names):

Earliest Priority Filing Date: 3-16-00

***For Sequence Searches Only* Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.**

Please search therapeutic method of attached claims. Active agent can be the carbohydrate (15-24) or an antibody (25-26).

Thanks.
Kathleen

RECEIVED FEB 18 2003 Technology Center (STIC)

Jan Delaval
Reference Librarian
Biotechnology & Chemical Library
CM1 1E07 – 703-308-4498
jan.delaval@uspto.gov

****

STAFF USE ONLY

Searcher: [illegible]
Searcher Phone #: 4498
Searcher Location:
Date Searcher Picked Up: 3/12/03
Date Completed: 3/12/03
Searcher Prep & Review Time:
Clerical Prep Time: 20
Online Time: + 120

Type of Search

NA Sequence (#)
AA Sequence (#)
Structure (#)
Bibliographic ✓
Litigation
Fulltext
Patent Family
Other

Vendors and cost where applicable

STN ✓
Dialog
Questel/Orbit
Dr.Link
Lexis/Nexis
Sequence Systems
WWW/Internet
Other (specify)

PTO-1590 (8-01)

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=> fil reg
FILE 'REGISTRY' ENTERED AT 10:08:42 ON 12 MAR 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:   11 MAR 2003  HIGHEST RN 497913-82-3
DICTIONARY FILE UPDATES:  11 MAR 2003  HIGHEST RN 497913-82-3

TSCA INFORMATION NOW CURRENT THROUGH MAY 20, 2002

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

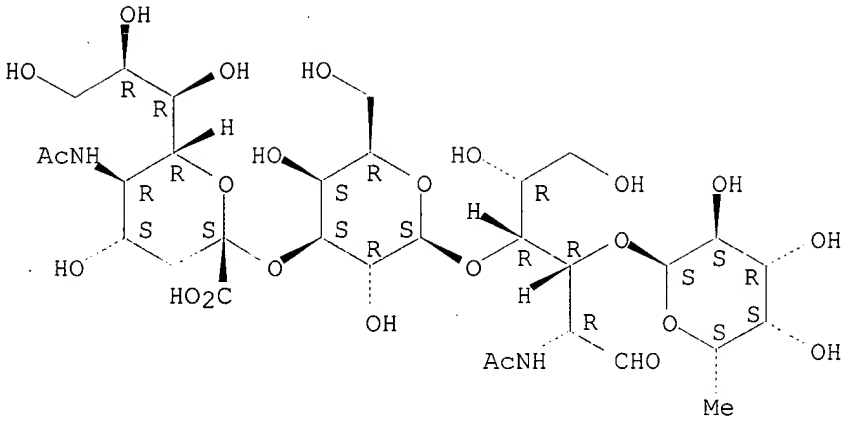
Experimental and calculated property data are now available.  See HELP
PROPERTIES for more information.  See STNote 27, Searching Properties
in the CAS Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> d ide can tot l1

L1   ANSWER 1 OF 2  REGISTRY  COPYRIGHT 2003 ACS
RN   98603-84-0  REGISTRY
CN   D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-
     galactopyranosyl-(1.fwdarw.4)-O-[6-deoxy-.alpha.-L-galactopyranosyl-
     (1.fwdarw.3)]-2-(acetylamino)-2-deoxy- (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   3'-Sialyl-Lewis X
CN   Sialyl Lex tri
CN   Sialyl-Lewis X
CN   SLex
CN   SSEA 1
FS   STEREOSEARCH
DR   149655-51-6
MF   C31 H52 N2 O23
SR   CA
LC   STN Files:   ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, CA, CAPLUS,
       CASREACT, CEN, CHEMCATS, CIN, CSCHEM, PROMT, TOXCENTER, USPATFULL

Absolute stereochemistry.  Rotation (+).
```

Jan Delaval
Reference Librarian
Biotechnology & Chemical Library
CM1 1E07 – 703-308-4498
jan.delaval@uspto.gov



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

341 REFERENCES IN FILE CA (1962 TO DATE)
61 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
345 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:131152

REFERENCE 2: 138:121274

REFERENCE 3: 138:53590

REFERENCE 4: 138:51620

REFERENCE 5: 138:49517

REFERENCE 6: 138:44697

REFERENCE 7: 138:3622

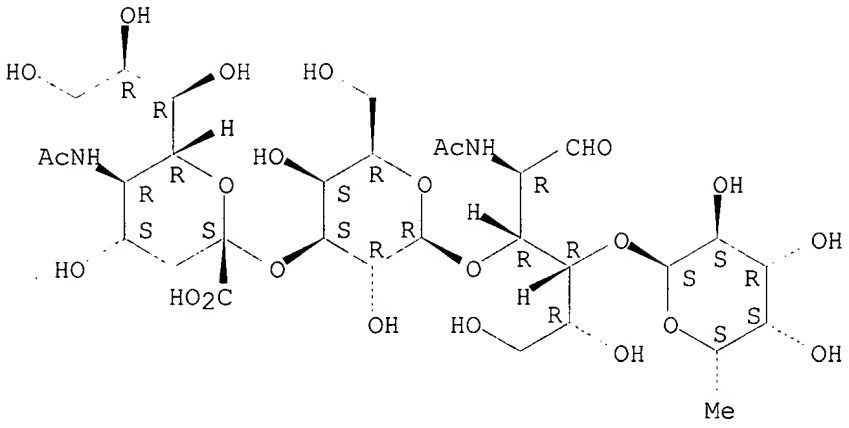
REFERENCE 8: 137:358087

REFERENCE 9: 137:357971

REFERENCE 10: 137:309114

L1 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2003 ACS
RN **92448-22-1** REGISTRY
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-[6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)]-2-(acetylamino)-2-deoxy- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 3'-Sialyl Lewis A
CN Sialyl Lea tri
CN Sialyl Lewis a
CN SLea
FS STEREOSEARCH
MF C31 H52 N2 O23
LC STN Files: BIOSIS, CA, CAPLUS, CHEMCATS, CSCHEM, PROMT, TOXCENTER, USPATFULL

Absolute stereochemistry. Rotation (-).



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

95 REFERENCES IN FILE CA (1962 TO DATE)
13 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
96 REFERENCES IN FILE CAPLUS (1962 TO DATE)

REFERENCE 1: 138:51620

REFERENCE 2: 138:49517

REFERENCE 3: 138:34960

REFERENCE 4: 137:292455

REFERENCE 5: 137:259076

REFERENCE 6: 137:214498

REFERENCE 7: 137:183288

REFERENCE 8: 137:149337

REFERENCE 9: 137:138368

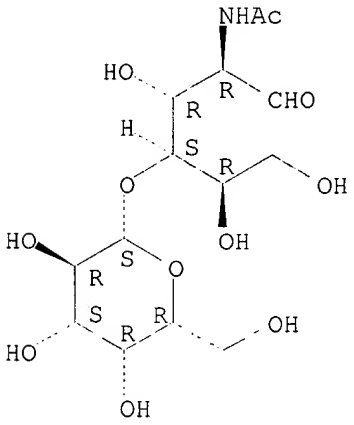
REFERENCE 10: 137:59509

=> d ide can l24

L24 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN **32181-59-2** REGISTRY
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN D-Glucosamine, N-acetyl-4-O-.beta.-D-galactopyranosyl- (6CI)
CN D-Glucose, 2-acetamido-2-deoxy-4-O-.beta.-D-galactopyranosyl- (7CI, 8CI)
OTHER NAMES:
CN 2-Acetamido-2-deoxy-4-O-.beta.-D-galactopyranosyl-D-glucose
CN Lactosamine, N-acetyl-
CN N-Acetyl-4-O-.beta.-D-galactopyranosyl-D-glucosamine
CN N-Acetyllactosamine
CN O-.beta.-D-Galactopyranosyl-(1.fwdarw.4)-2-deoxy-2-acetamido-D-glucose
AR 4307-58-8
FS STEREOSEARCH

DR 133432-89-0, 98529-93-2
MF C14 H25 N O11
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, MEDLINE, MSDS-OHS, PROMT, TOXCENTER, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

689 REFERENCES IN FILE CA (1962 TO DATE)
74 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
692 REFERENCES IN FILE CAPLUS (1962 TO DATE)
38 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

REFERENCE 1: 138:85196

REFERENCE 2: 138:85137

REFERENCE 3: 138:83382

REFERENCE 4: 138:83381

REFERENCE 5: 138:68701

REFERENCE 6: 138:54589

REFERENCE 7: 138:12748

REFERENCE 8: 138:4757

REFERENCE 9: 138:3756

REFERENCE 10: 138:1667

=> d his

(FILE 'HOME' ENTERED AT 08:49:44 ON 12 MAR 2003)
SET COST OFF

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     FILE 'REGISTRY' ENTERED AT 08:50:01 ON 12 MAR 2003
L1            2 S 92448-22-1 OR 98603-84-0
L2            0 S (92448-22-1 OR 98603-84-0)/CRN

     FILE 'HCAPLUS' ENTERED AT 08:59:17 ON 12 MAR 2003
L3          374 S L1
L4         1450 S SLEX OR SLEA OR SLEWX OR SLEWA OR (SLEW OR SLEWIS)()(X OR A)
L5            9 S SA()(LEX OR LEA OR (LEW OR LEWIS)()(X OR A))
L6            5 S SIAL? ACID()(LEX OR LEA OR (LEW OR LEWIS)()(X OR A))
L7          474 S SIAL?()(LEWISX OR LEWISA)
L8            3 S SIALYLEX OR SIALYLEA OR SIALYLLEWISX OR SIALYLLEWISA OR SIALY
L9         1707 S L3-L8
                E TENEBERG S/AU
L10          57 S E3,E4
                E HAMMARSTROM L/AU
L11         106 S E3-E8,E17,E18
                E HAMMARSTROEM L/AU
L12          93 S E3-E5,E14
                E KARLSSON K/AU
L13         325 S E3,E4,E17-E20
                E BOREN T/AU
L14          36 S E3-E5
                E BOEREN T/AU
L15           8 S L9 AND L10-L14
                E WO2000-SE514/AP,RPN
L16           1 S E3
                E SE99-1007/AP,PRN
L17           1 S E4
L18           1 S L16,L17 AND L3-L15
                SEL RN

     FILE 'REGISTRY' ENTERED AT 09:07:00 ON 12 MAR 2003
L19          27 S E1-E27
L20           9 S L19 AND OC5/ES
L21          18 S L19 NOT L20
L22          10 S L21 AND CERAMIDE
L23          19 S L20,L22
L24           1 S 32181-59-2

     FILE 'HCAPLUS' ENTERED AT 09:10:52 ON 12 MAR 2003
L25         696 S L24
L26        1327 S N()(ACETYLLACTOSAMINE OR ACETYL LACTOSAMINE)
L27          16 S L10-L15 AND L25,L26
L28          23 S L15-L18,L27
L29          22 S L28 NOT L18
                SEL RN

     FILE 'REGISTRY' ENTERED AT 09:13:35 ON 12 MAR 2003
L30         175 S E28-E202
L31         165 S L30 NOT L19
L32         164 S L31 NOT L1
L33          70 S L32 AND OC5/ES
L34          86 S L32 AND UNSPECIFIED
L35          75 S L34 NOT SQL/FA
L36          66 S L35 AND CERAMIDE
L37           9 S L35 NOT L36
L38          76 S L22,L36
                E CERAMIDE
L39        1565 S E3
L40        1375 S L39 NOT SQL/FA
L41        1346 S L40 AND UNSPECIFIED
L42          29 S L40 NOT L41
L43           4 S L42 AND OC5/ES
```

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L44          79 S L41 NOT MAN/CI
L45          73 S L44 NOT (MXS/CI OR COMPD OR WITH)
L46           6 S L44 NOT L45
L47        1263 S L41 AND 1/NC
L48          83 S L41 NOT L47
L49           4 S L48 NOT L42-L46
L50          20 S L34 NOT L36
L51          18 S L23 NOT L1,L24

     FILE 'HCAPLUS' ENTERED AT 09:27:19 ON 12 MAR 2003

     FILE 'REGISTRY' ENTERED AT 09:27:28 ON 12 MAR 2003

     FILE 'HCAPLUS' ENTERED AT 09:32:20 ON 12 MAR 2003
                E BLOOD-GROUP SUBSTANCES/CT
L52        1644 S E17-E23
                E E3+ALL
L53        1738 S E3(L)(LE OR LEA OR LEX OR LEW? OR SIAL?)
L54         279 S E3 (L) FUCOS?
L55          22 S L10-L15 AND L52-L54
L56        4477 S L9,L25,L26,L52-L54
                E HELICOP/CT
                E HELICOB/CT
L57        5084 S E28-E29
                E E28+ALL
L58        6293 S E6,E5+NT
L59        7533 S E5/BI OR E6/BI OR E7/BI OR E8/BI
L60        7666 S (H OR C OR HELICOBACT? OR CAMPYLOBACT?)()PYLORI?
L61         116 S L56 AND L57-L60
                E ADHESINS/CT
                E E3+ALL
L62          27 S L56 AND E4,E5,E3+NT
                E E10+ALL
L63         180 S L56 AND E2+NT
L64         261 S L56 AND E1+NT
                E EPITHELIUM/CT
                E E20+ALL
L65         925 S E2
                E EPITHELIUM/CT
                E E22+ALL
L66         146 S E2
                E EPITHELIUM/CT
                E E30+ALL
L67        5644 S E2
L68         209 S E4
                E EPITHELIUM/CT
                E E53+ALL
L69        1158 S E2
                E EPITHELIUM/CT
                E E59+ALL
L70          53 S E2

     FILE 'REGISTRY' ENTERED AT 09:44:26 ON 12 MAR 2003
                E EPITHELIUM SMALL INTESTINE/CN

     FILE 'HCAPLUS' ENTERED AT 09:44:26 ON 12 MAR 2003
                E EPITHELIUM SMALL INTESTINE/CT
                E E3+ALL
L71         659 S E2
                E EPITHELIUM SMALL INTESTINE/CT
                E GASTRIC MUCOSA/CT
                E E3+ALL
L72        7298 S E2
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L73          101 S E10
L74           67 S L56 AND L65-L73
                 E DIGESTIVE TRACT/CT
                 E E3+ALL
L75          741 S E3+NT AND L56
                 E DIGESTIVE TRACT/CT
                 E ULCER/CT
L76         2089 S E5,E7,E8,E10
L77          290 S E15,E16,E17,E18
                 E E3+ALL
L78         9575 S E3,E2
                 E E4+ALL
L79         5828 S E4,E3,E8-E11
L80          749 S L56 AND L75-L79
L81           62 S L61 AND L62-L64,L74,L80
L82           14 S L81 AND ?FUCO?
L83           39 S L61 AND ?FUCO?
L84           39 S L82,L83
L85           30 S L84 AND (PD<=20000316 OR PRD<=20000316 OR AD<=20000316)
L86            9 S L84 NOT L85
L87           23 S L28,L29
L88           40 S L55,L87
L89           40 S L88 AND L56
L90           23 S L89 AND L57-L84
L91           17 S L89 NOT L90
L92           46 S L85,L90
L93           40 S L92 AND (PD<=20000316 OR PRD<=20000316 OR AD<=20000316)
L94           23 S L92 AND L10-L14
L95           23 S L93 NOT L94
L96          357 S L25,L26 (L) FUCO?
L97           14 S L96 AND L57-L60
L98            2 S L96 AND PHARMACEUT?/SC,SX
L99           16 S L96 AND PHARMACOL?/SC,SX
L100          17 S L98,L99
L101          24 S L25,L26 (L) THU/RL
L102          23 S L101 NOT L97-L100
                 SEL DN AN 1 4
L103           2 S E1-E6
L104          23 S L94,L103

     FILE 'REGISTRY' ENTERED AT 10:08:42 ON 12 MAR 2003

=> fil hcaplus
FILE 'HCAPLUS' ENTERED AT 10:09:00 ON 12 MAR 2003
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FILE COVERS 1907 - 12 Mar 2003  VOL 138 ISS 11
FILE LAST UPDATED: 11 Mar 2003  (20030311/ED)

 This file contains CAS Registry Numbers for easy and accurate
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substance identification.

=> d l104 all hitstr tot

L104 ANSWER 1 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN **2003:22694** HCAPLUS
DN **138:83382**
TI Polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases
IN Natunen, Jari; Miller-Podraza, Halina; **Teneberg, Susann**; Angstroem, Jonas; **Karlsson, Karl-Anders**
PA Carbion Oy, Finland
SO PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DT Patent
LA English
IC A61K031-702; A61K031-722; A61K031-727
CC 1-9 (Pharmacology)
Section cross-reference(s): 63
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003002128	A1	20030109	WO 2002-FI575	20020628
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	FI 2001001403	A	20021230	FI 2001-1403	20010629
	WO 2002056893	A1	20020725	WO 2002-FI43	20020118
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	FI 2001-1403	A	20010629		
	WO 2002-FI43	A	20020118		
	FI 2001-118	A	20010119		

AB The present invention relates to a compn. comprising a polysaccharide with **Helicobacter pylori** receptor activity and, optionally, an oligosaccharide receptor of **Helicobacter pylori** or an analog or a deriv. thereof and/or a gastric epithelium protecting compd. for use in the treatment or prophylaxis of any condition due to the presence of **Helicobacter pylori**. Binding assays revealed the isoreceptors and specificity of binding of glycolipids such as Neu5Gc.alpha.3Gal.beta.4GlcNAc.beta.3Gal.beta.4GlcNAc.beta.3Gal.beta.4Glc.beta.Cer.
ST polysaccharide Helicobacteri receptor activity gastric disease
IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Helicobacter pylori**; polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)
IT **Helicobacter pylori**

Stomach, disease
(polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)

IT Glycolipids
Polysaccharides, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)

IT 63-42-3 5965-66-2 13007-32-4 14116-68-8 **32181-59-2** 32694-82-9 35259-23-5 35960-33-9 41744-59-6 47491-70-3 50787-09-2 56573-54-7 62897-09-0 71012-19-6 71833-54-0 71833-57-3 71950-01-1 71950-33-9, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 72067-19-7 72711-52-5 73201-40-8 73379-94-9, Ceramide, 1-O-[O-[N-(hydroxyacetyl)-.alpha.-neuraminosyl-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 73467-80-8 75034-76-3, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 75598-07-1 75645-24-8 75645-25-9 75645-27-1 77356-46-8 77538-29-5, Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-.beta.-D-galactopyranosyl-(1.fwdarw.3)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 77538-32-0 78990-73-5 80619-72-3 82030-41-9 83563-61-5 86993-34-2 87856-44-8 88161-63-1, Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 92448-21-0 95210-85-8 95896-53-0 96638-04-9, Ceramide, 1-O-[O-.alpha.-D-galactopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 97666-64-3 99147-61-2 99147-62-3 101627-01-4 106828-82-4, Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-[.alpha.-D-galactopyranosyl-(1.fwdarw.3)]-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 110540-11-9 114643-66-2 138398-63-7 151183-78-7, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 153366-25-7 186467-26-5, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.6)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 189201-22-7, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-amino-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 222540-52-5, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-amino-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 289719-54-6 443660-37-5 443660-39-7 443660-41-1 443660-43-3 443660-58-0 443660-60-4 443660-62-6 443660-64-8 443660-66-0

443660-68-2 443660-70-6 443660-72-8 443660-78-4 443660-80-8
443660-83-1 443660-85-3 443660-87-5 443660-90-0 443660-94-4
443660-98-8 443661-01-6 482373-64-8 482373-65-9 482620-51-9,
Ceramide, 1-O-[O-.alpha.-D-galactopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-amino-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 482626-85-7, Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-.alpha.-D-galactopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]-
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)

IT 9004-61-9, Hyaluronic acid 9007-27-6, Chondroitin 9007-27-6D, Chondroitin, **fucosylated** 9007-28-7, Chondroitin sulfate 9012-76-4, Chitosan
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)

RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD

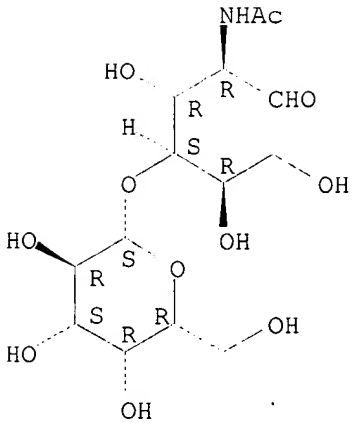
RE

(1) A Science Invest Ab; WO 0143751 A1 2001 HCAPLUS
(2) Alberta Research Council; WO 9303735 A1 1993 HCAPLUS
(3) Boren, T; WO 0056343 A1 2000 HCAPLUS
(4) Ghen Corporation; EP 1002805 A1 2000 HCAPLUS
(5) Goldman; US 5204118 A 1993 HCAPLUS
(6) Kabushiki Kaisha Yakult Honshaminato-Ku; EP 1120100 A1 2001 HCAPLUS
(7) Krivan; US 5386027 A 1995 HCAPLUS
(8) Masayuki, M; Cancer Research 1989, V49, P5689
(9) Monteiro, M; The Journal of Biological Chemistry 1998, V273(19), P11533 HCAPLUS
(10) Mysore, J; Gatroenterology 1999, V117, P1316 MEDLINE
(11) Neose Technoligies Inc; WO 9741875 A1 1997 HCAPLUS
(12) Roland, R; Journal of Immunology 2000, V168, P3033
(13) Spilburg; US 5679375 A 1997 HCAPLUS
(14) Syntek Ab; WO 8603971 A1 1986 HCAPLUS
(15) Veerman, E; Glycobiology 1997, V7(6), P737 HCAPLUS
(16) Yamada; US 5468503 A 1995 HCAPLUS
(17) Zopf; US 5753630 A 1998 HCAPLUS

IT **32181-59-2**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(polysaccharides with **Helicobacter pylori** receptor activity for treatment of gastric diseases)

RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 2 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2003:22693 HCAPLUS
DN 138:83381
TI Glycosidase inhibitors for treatment of gastric disease.
IN Natunen, Jari; Miller-Podraza, Halina; **Teneberg, Susann**; Angstroem, Jonas; **Karlsson, Karl-Anders**
PA Carbion Oy, Finland
SO PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-702
ICS A61P001-04; A61P031-04
CC 1-9 (Pharmacology)
Section cross-reference(s): 63
FAN.CNT 3

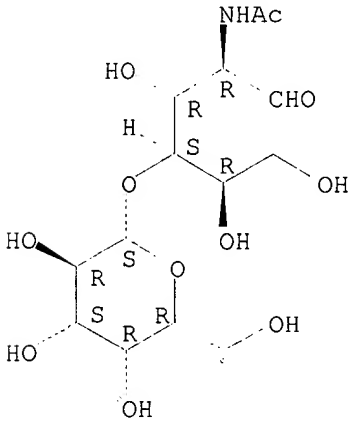
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003002127	A1	20030109	WO 2002-FI574	20020628
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	FI 2001001402	A	20021230	FI 2001-1402	20010629
	FI 2001001403	A	20021230	FI 2001-1403	20010629
PRAI	FI 2001-1402	A	20010629		
	FI 2001-1403	A	20010629		

AB The present invention relates to the use of a glycosidase inhibitor for the manuf. of a medicament for the treatment of a disease, wherein glycosidase enzymes hydrolyze glycoconjugates of a patient to reveal neutral glycan receptors of an pathogenic agent, and wherein the revealed neutral glycan receptor comprise a oligosaccharide sequence.
ST glycosidase inhibitor gastric disease; polysaccharide glycosidase inhibitor gastric disease
IT Anti-infective agents
Helicobacter pylori
Human
Stomach, disease

(glycosidase inhibitors for treatment of gastric disease)
IT Glycolipids
Polysaccharides, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycosidase inhibitors for treatment of gastric disease)
IT 9032-92-2, Glycosidase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(glycosidase inhibitors for treatment of gastric disease)
IT 35960-33-9 56573-54-7 71012-19-6 71833-54-0 71833-57-3 71950-01-1 72067-19-7 72412-78-3, Ceramide, 1-O-[O-2-amino-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 72711-52-5 73201-40-8 73467-80-8 77538-29-5, Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-.beta.-D-galactopyranosyl-(1.fwdarw.3)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 77538-32-0 78990-73-5 80619-72-3 82030-41-9 86993-34-2 88161-63-1, Ceramide, 1-O-(O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 97666-64-3 99147-61-2 99147-62-3 106828-82-4, Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-[.alpha.-D-galactopyranosyl-(1.fwdarw.3)]-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 110540-11-9 186467-26-5, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.6)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 189201-22-7, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-amino-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 222540-52-5, Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-amino-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- 482620-51-9 482626-85-7 482628-99-9 482629-00-5
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycosidase inhibitors for treatment of gastric disease)
IT 63-42-3 13007-32-4 14116-68-8 **32181-59-2** 41744-59-6 50787-09-2 54832-51-8 62897-09-0 66580-68-5 75645-24-8 75645-25-9 75645-27-1 77356-46-8 87856-44-8 138398-63-7
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycosidase inhibitors for treatment of gastric disease)
RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) A Science Invest Ab; WO 0143751 A1 2001 HCAPLUS
(2) Boren, T; WO 0056343 A1 2000 HCAPLUS
(3) Holmes, E; Archives of Biochemistry Biophysics 1990, V277, P181 HCAPLUS
(4) Jimenez; US 5242800 A 1993 HCAPLUS
(5) Kalsson; US 4859769 A 1989 HCAPLUS
(6) Kim, C; Antiviral Chemistry & Chemotherapy 1999, V10, P141 HCAPLUS
(7) Krivan; US 5217715 A 1993 HCAPLUS
(8) Krivan; US 5386027 A 1995 HCAPLUS
(9) Maan-Abul-Milh; Glycoconjugate Journal 2001, V18, P253
(10) Masayuki, M; Cancer Research 1989, V49, P5689
(11) McNicholl, I; The Annals of Pharmacotherapy 2001, V35, P57 HCAPLUS
(12) Miller-Podraza, H; Infection and Immunity 1997, V65(6), P2480 HCAPLUS
(13) Neose Technologies Inc; WO 9741875 A1 1997 HCAPLUS

(14) Normark, J; WO 9418986 A1 1994 HCAPLUS
(15) Per, F; J Biochem 1990, V108, P466
(16) Simon, P; Infection and Immunity 1997, V65(2), P750 HCAPLUS
(17) Toepfer; US 6136790 A 2000 HCAPLUS
(18) Zopf; US 5514660 A 1996 HCAPLUS
IT **32181-59-2**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(glycosidase inhibitors for treatment of gastric disease)
RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI)
(CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 3 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2002:658147 HCAPLUS
DN 137:198237
TI Potential use of **Helicobacter pylori** sialic acid
binding adhesin gene in diagnosis and treatment of infection
IN **Boren, Thomas; Hammarstroem, Lennart**
PA Swed.
SO PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K014-205
ICS A61K039-106
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 3, 6, 14
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002066502	A1	20020829	WO 2002-SE301	20020221

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-269889P P 20010221
AB An isolated **Helicobacter pylori** protein binding to

sialyl-Lewis x antigen and having an approx. mol. wt. of 66kDa and **sialyl-Lewis x** antigen-binding **H.pylori** alleles of the protein, recombinant forms of the protein or the protein alleles, and **sialyl-Lewis x** antigen binding portions of the proteins, are disclosed. The protein or portion of protein maybe used as a medicament or diagnostic antigen, and can be used in a method of detg. the presence of **sialyl-Lewis x** antigen-binding **H.pylori** bacteria in a biol. sample. Further, a DNA mol. encoding the protein or portion of protein, a vector comprising the DNA mol., and a host transformed with the vector are comprised by the disclosure. Addnl., a method of detg. the presence of **sialyl-Lewis x** or related carbohydrate structures in a sample, is described. This method has a wide range of different applications.

ST Helicobacter sialic acid binding adhesin sequence; diagnosis treatment Helicobacter infection sabA gene

IT Molecular weight
(66 kDa, of sialic acid binding adhesin; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT **Blood-group substances**
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(**Lex, sialyl**, SABA protein binding to, detection of; potential use of **Helicobacter pylori** **sialic** acid binding adhesin gene in diagnosis and treatment of infection)

IT Carbohydrates, biological studies
RL: ANT (Analyte); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study)
(SabA protein in detection of; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Diagnosis
(mol.; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(monoclonal, to SabA protein; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Protein sequences
(of SabA protein of **Helicobacter pylori**; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Molecular association
(of sialic acid binding adhesin to **sialyl-Lewis x** antigen; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT **Helicobacter pylori**
Molecular cloning
(potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Gene, microbial
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sabA, of **Helicobacter pylori**; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT **Adhesins**

RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sialic acid binding, sabA gene for; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Alleles
(**sialyl-Lewis x** antigen-binding, of **Helicobacter pylori**; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(to SabA protein; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT 452897-15-3 452897-16-4 452897-17-5 452897-18-6
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(SabA peptide sequence; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT 452984-94-0
RL: PRP (Properties)
(Unclaimed; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT 452984-59-7, Adhesin (**Helicobacter pylori** gene sabA)
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

IT 452984-95-1 452984-96-2 452984-97-3 452984-98-4 452984-99-5 452985-00-1 452985-01-2
RL: PRP (Properties)
(unclaimed nucleotide sequence; potential use of **Helicobacter pylori** sialic acid binding adhesin gene in diagnosis and treatment of infection)

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Alm, R; Infection and Immunity 2000, V68(7), P4155 HCAPLUS
(2) Alm, R; Nature 1999, V397, P176 HCAPLUS
(3) Astra, A; WO 9824475 A1 1998 HCAPLUS
(4) Boren, T; WO 9747646 A1 1997 HCAPLUS
(5) Boren, T; WO 0056343 A1 2000 HCAPLUS
(6) Dag, I; SCINECE 1998, V279, P373
(7) Merieux Oravax Societe En Nom Collectif; WO 9843479 A1 1998 HCAPLUS

L104 ANSWER 4 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 2002:571795 HCAPLUS

DN 137:261037

TI **Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation

AU Mahdavi, Jafar; Sonden, Berit; Hurtig, Marina; Olfat, Farzad O.; Forsberg, Lina; Roche, Niamh; Angstrom, Jonas; Larsson, Thomas; **Teneberg, Susann; Karlsson, Karl-Anders**; Attraja, Siiri; Wadstroem, Torkel; Kersulyte, Dangeruta; Berg, Douglas E.; Dubois, Andre; Petersson, Christoffer; Magnusson, Karl-Eric; Norberg, Thomas; Lindh, Frank; Lundskog, Bertil B.; Arnqvist, Anna; **Hammarstroem, Lennart; Boren, Thomas**

CS Department of Odontology/Oral Microbiology, Umea University, Umea, SE-901 87, Swed.

SO Science (Washington, DC, United States) (2002), 297(5581), 573-578
CODEN: SCIEAS; ISSN: 0036-8075
PB American Association for the Advancement of Science
DT Journal
LA English
CC 14-3 (Mammalian Pathological Biochemistry)

AB **Helicobacter pylori** adherence in the human gastric mucosa involves specific bacterial adhesins and cognate host receptors. Here, the authors identify sialyl-dimeric-Lewis x glycosphingolipid as a receptor for **H. pylori** and show that **H. pylori** infection induced formation of **sialyl-Lewis x** antigens in gastric epithelium in humans and in a Rhesus monkey. The corresponding sialic acid-binding adhesin (SabA) was isolated with the "retagging" method, and the underlying SabA gene (JHP662/HP0725) was identified. The ability of many **H. pylori** strains to adhere to sialylated glycoconjugates expressed during chronic inflammation might thus contribute to virulence and the extraordinary chronicity of **H. pylori** infection.

ST SabA adhesin Helicobacter infection inflammation stomach

IT **Adhesion, biological**
Helicobacter pylori
Human
Virulence (microbial)
(**Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Lex, sialyl; Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

IT **Adhesins**
Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(SabA; **Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

IT Inflammation
(chronic; **Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

IT **Stomach**
(**epithelium; Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

IT **Stomach, disease**
(infection; **Helicobacter pylori** SabA adhesin in persistent infection and chronic inflammation)

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Akopyants, N; Mol Microbiol 1998, V28, P37 HCAPLUS
(2) Alm, R; Nature 1999, V397, P176 HCAPLUS
(3) Alper, J; Science 2001, V291, P2338 HCAPLUS
(4) Amado, M; Gastroenterology 1998, V114, P462 MEDLINE
(5) Arnqvist, A; in preparation
(6) Boren, T; Science 1993, V262, P1892 HCAPLUS
(7) Boren, T; Science 1994, V264, P1387
(8) Censini, S; Proc Natl Acad Sci USA 1996, V93, P14648 HCAPLUS
(9) Clausen, H; Vox Sang 1989, V56, P1 HCAPLUS
(10) Cover, T; Principles of Bacterial Pathogenesis 2001, P509 HCAPLUS
(11) Dubois, A; Gastroenterology 1999, V116, P90 MEDLINE
(12) Falk, P; Proc Natl Acad Sci USA 1993, V90, P2035 HCAPLUS
(13) Falk, P; Proc Natl Acad Sci USA 1995, V92, P1515 HCAPLUS
(14) Gerhard, M; Helicobacter pylori: Molecular and Cellular Biology 2001
(15) Gerhard, M; Proc Natl Acad Sci USA 1999, V96, P12778 HCAPLUS
(16) Guruge, J; Proc Natl Acad Sci USA 1998, V95, P3925 HCAPLUS
(17) Hakomori, S; Gangliosides and Cancer 1989, P58
(18) Herron, M; Science 2000, V288, P1653 HCAPLUS

(19) Hurtig, M; in preparation
(20) Ilver, D; Science 1998, V279, P373 HCAPLUS
(21) Karlsson, K; Mol Microbiol 1998, V29, P1 HCAPLUS
(22) Madrid, J; Histochemistry 1990, V95, P179 HCAPLUS
(23) Magnani, J; Science 1981, V212, P55 HCAPLUS
(24) Ota, H; Virchows Arch 1998, V433, P419 HCAPLUS
(25) Sakamoto, J; Cancer Res 1989, V49, P745 HCAPLUS
(26) Scatchard, G; Ann NY Acad Sci 1949, V51, P660 HCAPLUS
(27) Segal, E; Proc Natl Acad Sci USA 1999, V96, P14559 HCAPLUS
(28) Sipponen, P; Acta Pathol Microbiol Immunol Scand 1986, V94, P305 MEDLINE
(29) Sipponen, P; Scand J Gastroenterol 1989, V24, P581 MEDLINE
(30) Syder, A; Mol Cell 1999, V3, P263 HCAPLUS
(31) Tomb, J; Nature 1997, V388, P539 HCAPLUS

L104 ANSWER 5 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN **2002:555358** HCAPLUS
DN **137:114486**
TI Novel receptors for **Helicobacter pylori** and use thereof
IN Miller-Podraza, Halina; **Teneberg, Susann**; Angstroem, Jonas; **Karlsson, Karl-Anders**; Natunen, Jari
PA Carbion Oy, Finland
SO PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-702
ICS C07H015-04; C07H003-06; A61P001-04; A61P031-04
CC 63-3 (Pharmaceuticals)
Section cross-reference(s): 9, 15, 17, 33
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002056893	A1	20020725	WO 2002-FI43	20020118
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	FI 2001000118	A	20020720	FI 2001-118	20010119
	WO 2003002128	A1	20030109	WO 2002-FI575	20020628
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	FI 2001-118	A	20010119		
	FI 2001-1403	A	20010629		
	WO 2002-FI43	A	20020118		

AB The present invention describes a substance or a receptor comprising **Helicobacter pylori**-binding oligosaccharide sequence [Gal(A)q(NAc)r/Glc(A)q(NAc)r.alpha.3/.beta.3]s[Gal.beta.4GlcNAc.beta.3]tGal.beta.4Glc(NAc)u wherein q, r, s, t, and u are each independently 0 or 1, and the use thereof in, e.g., pharmaceutical and nutritional compns. for the treatment of conditions due to the presence of **Helicobacter**

pylori. The invention is also directed to the use of the receptor for diagnostics of **Helicobacter pylori**.

ST Helicobacter receptor oligosaccharide sequence

IT **Digestive tract**
(**H. pylori** presence in; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Stomach, neoplasm**
(adenocarcinoma; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Diagnosis
(agents; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Stomach, disease**
(autoimmune gastritis; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Adhesins**
RL: ANT (Analyte); ANST (Analytical study)
(bacterial; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Bacteria (Eubacteria)
Virus
(binding of; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Toxins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(binding of; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Drug delivery systems
(carriers; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Polysaccharides, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(conjugates; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Intestine, disease**
(duodenum, ulcer; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Stomach, disease**
(gastritis, chronic superficial; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Milk substitutes
(human; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Lymphoma
(non-Hodgkin's; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Anti-inflammatory agents
(nonsteroidal, -related stomach injury; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Autoimmune disease
Diagnosis
Heart, disease
Helicobacter pylori
Human
Liver, disease
Micelles

Pancreas, disease
Skin, disease
Test kits
Vaccines
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Oligosaccharides, biological studies
Receptors
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Glycosphingolipids
RL: PNU (Preparation, unclassified); PUR (Purification or recovery); PREP (Preparation)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Glycolipids
RL: PUR (Purification or recovery); PREP (Preparation)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Antibiotics
(oligosaccharide conjugates; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Anemia (disease)
(pernicious anemia; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Death
(sudden infant death syndrome; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Diet
(supplements; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT Clostridium difficile
(toxin of; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT **Stomach, disease**
(ulcer; novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT 9031-11-2, .beta.-Galactosidase 105503-61-5
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT 13007-32-4P, Lacto-N-neotetraose **32181-59-2P** 32694-82-9P
62897-09-0P 64309-00-8P, P-Lacto-N-neohexaose 75645-27-1P
87856-44-8P 95210-85-8P 95896-53-0P 96623-71-1P 97604-31-4P
136247-80-8P 138398-63-7P 178177-03-2P 289719-54-6P 443660-37-5P
443660-39-7P 443660-41-1P 443660-43-3P 443660-47-7P 443660-49-9P
443660-52-4P 443660-54-6P 443660-56-8P 443660-58-0P 443660-60-4P
443660-62-6P 443660-64-8P 443660-66-0P 443660-68-2P 443660-70-6P
443660-72-8P 443660-78-4P 443660-80-8P 443660-83-1P 443660-85-3P
443660-87-5P 443660-90-0P 443660-94-4P 443660-98-8P 443661-01-6P
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PAC (Pharmacological activity); PNU (Preparation, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

IT 1406-05-9D, Penicillin, oligosaccharide conjugates
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

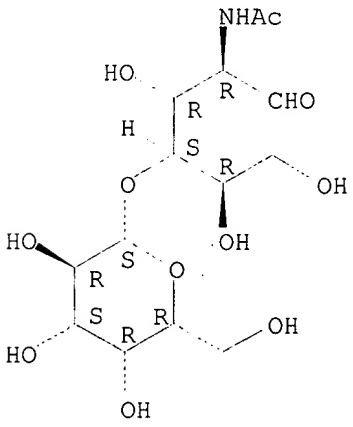
(1) A Science Invest Ab; WO 0143751 A1 2001 HCAPLUS
(2) Abul-Milh, M; Glycoconjagate Journal 2001, V18, P253 HCAPLUS
(3) Biocarb Ab; EP 0098252 B1 1984 HCAPLUS
(4) Gold, B; Infection and Immunity 1993, P2632 HCAPLUS
(5) Holmes, E; Archives of Biochemistry and Biophysics 1990, V277(1), P181 HCAPLUS
(6) Jonas, A; Glycobiology 1998, V8(4), P297
(7) Miller-Podraza, H; Infection and Immunity 1997, V65(6), P2480 HCAPLUS
(8) Murakami, M; JP A10045602 1998
(9) Per, F; J Biochem 1990, V108, P466
(10) Svenska Sockerfabriks Ab; EP 0089938 A1 1983 HCAPLUS
(11) Syntek Ab; WO 8604065 A1 1986 HCAPLUS

IT **32181-59-2P**
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); PAC (Pharmacological activity); PNU (Preparation, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(novel oligosaccharide receptors for **Helicobacter pylori** and therapeutic and diagnostic uses thereof)

RN 32181-59-2 HCAPLUS

CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 6 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 2001:914807 HCAPLUS

DN 136:165626

TI Different glycosphingolipid composition in human neutrophil subcellular compartments

AU Karlsson, Anna; Miller-Podraza, Halina; Johansson, Petra; **Karlsson, Karl-Anders**; Dahlgren, Claes; **Teneberg, Susann**

CS Department of Medical Microbiology and Immunology, Goteborg University, Goteborg, 405 30, Swed.

SO Glycoconjugate Journal (2001), 18(3), 231-243
CODEN: GLJOEW; ISSN: 0282-0080

PB Kluwer Academic Publishers

DT Journal

LA English

CC 15-1 (Immunochemistry)

AB The binding of a no. of carbohydrate-recognizing ligands to glycosphingolipids and polyglycosylceramides of human neutrophil subcellular fractions (plasma membranes/secretory vesicles of resting and ionomycin-stimulated cells, specific and azurophil granules) was examd. using the chromatogram binding assay. Several organelle-related differences in glycosphingolipid content were obsd. The most prominent difference was a decreased content of the GM3 ganglioside in plasma membranes of activated neutrophils. Gangliosides recognized by anti-VIM-2 antibodies were detected mainly in the acid fractions of azurophil and specific granules. Slow-migrating gangliosides and polyglycosylceramides with **Helicobacter pylori**-binding activity were found in all acid fractions. A non-acid triglycosylceramide, recognized by Gal.alpha.4Gal-binding Escherichia coli, was detected in the plasma membrane/secretory vesicles but not in the azurophil and specific granules. Although no defined roles of glycosphingolipids have yet been conclusively established with respect to neutrophil function, the fact that many of the identified glycosphingolipids are stored in granules, is in agreement with their role as receptor structures that are exposed on the neutrophil cell surface upon fusion of granules with the plasma membrane. Accordingly, we show that neutrophil granules store specific carbohydrate epitopes that are upregulated to the plasma membrane upon cell activation.

ST glycosphingolipid polyglycosylceramide neutrophil cell membrane granule

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(**Lex, sialyl**; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(**Lex**; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Glycosphingolipids
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(acidic; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Neutrophil
(activation; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Cell membrane
Human
(different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Carbohydrates, biological studies
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Cell activation
(neutrophil; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Glycosphingolipids
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(non-acidic; different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Epitopes
(of different glycosphingolipid compn. in human neutrophil subcellular compartments)

IT Organelle
(secretory granule; different glycosphingolipid compn. in human

neutrophil subcellular compartments)
IT Cerebrosides
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(tri; different glycosphingolipid compn. in human neutrophil subcellular compartments)
IT 4682-48-8, Lactosylceramide 56573-54-7, Neolactotetraosylceramide 73467-80-8, Lactotriaosylceramide 86993-34-2, Neolactohexaosylceramide 89678-50-2, Ganglioside GM3
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(different glycosphingolipid compn. in human neutrophil subcellular compartments)
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Angstroem, J; Glycobiology 1998, V8, P297
(2) Anon; Inflammation Basic Principles and Clinical Correlates 1999
(3) Bainton, D; J Cell Biol 1966, V28, P277 MEDLINE
(4) Bainton, D; J Exp Med 1971, V143, P907
(5) Berton, G; Inflammation Basic Principles and Clinical Correlates 1999, P703
(6) Bock, K; J Biol Chem 1985, V260, P8545 HCAPLUS
(7) Boeyum, A; Scand J Lab Invest 1968, V21, P77
(8) Borregaard, N; Blood 1997, V89, P3503 HCAPLUS
(9) Borregaard, N; J Cell Biol 1983, V97, P52 HCAPLUS
(10) Bos, A; Biochem Biophys Acta 1978, V525, P37 HCAPLUS
(11) Ciopraga, J; J Biochem 2000, V128, P855 HCAPLUS
(12) Dahlgren, C; Biochem J 1995, V311, P667 HCAPLUS
(13) DeChatelet, L; Biochem Med 1970, V4, P61 HCAPLUS
(14) Falk, K; Arch Biochem Biophys 1979, V192, P164 HCAPLUS
(15) Falk, K; Arch Biochem Biophys 1979, V192, P177 HCAPLUS
(16) Falk, K; Arch Biochem Biophys 1979, V192, P191 HCAPLUS
(17) Fukushi, Y; J Biol Chem 1984, V259, P10511 HCAPLUS
(18) Gottlieb, C; J Hematol 1965, V25, P875 HCAPLUS
(19) Handa, K; Biochemistry 1997, V36, P12412 HCAPLUS
(20) Hansson, G; Anal Biochem 1985, V146, P158 HCAPLUS
(21) Karlsson, K; Annu Rev Biochem 1989, V58, P309 HCAPLUS
(22) Karlsson, K; Methods Enzymol 1987, V138, P212 HCAPLUS
(23) Kneip, B; J Leukocyte Biol 1998, V63, P83
(24) Koerner, T; Biochemistry 1983, V22, P2676 HCAPLUS
(25) Kornfeld, R; Annu Rev Biochem 1985, V54, P631 MEDLINE
(26) Macher, B; J Biol Chem 1980, V255, P2092 HCAPLUS
(27) Macher, B; J Biol Chem 1988, V263, P10186 HCAPLUS
(28) Magnani, J; Science 1981, V212, P55 HCAPLUS
(29) Miller-Podraza, H; Biochim Biophys Acta 1993, V1168, P330 HCAPLUS
(30) Miller-Podraza, H; Glycoconj J 1997, V14, P231 HCAPLUS
(31) Miller-Podraza, H; Infect Immun 1997, V65, P2480 HCAPLUS
(32) Miller-Podraza, H; Infect Immun 1999, V67, P6309 HCAPLUS
(33) Muething, J; Glycobiology 1996, V6, P147 HCAPLUS
(34) Samuelsson, B; Methods Enzymol 1990, V193, P623 HCAPLUS
(35) Stellner, K; Arch Biochem Biophys 1973, V155, P464 HCAPLUS
(36) Stroud, M; Biochemistry 1996, V35, P758 HCAPLUS
(37) Stroud, M; Biochemistry 1996, V35, P770 HCAPLUS
(38) Svanborg Eden, C; Infect Immun 1984, V44, P672 MEDLINE
(39) Symington, F; J Biol Chem 1987, V262, P11356 HCAPLUS
(40) Symington, F; J Immunol 1989, V142, P2784 HCAPLUS
(41) Teneberg, S; J Biol Chem 1994, V269, P8554 HCAPLUS
(42) Teneberg, S; J Biol Chem 1997, V272, P19067 HCAPLUS
(43) Tewari, R; Infect Immun 1994, V62, P5296 HCAPLUS
(44) Thorn, J; Biochemistry 1992, V31, P6509 HCAPLUS
(45) Waldi, D; Duennschicht-Chromatographie 1962, P496
(46) Yang, H; J Biol Chem 1971, V246, P1192 MEDLINE

L104 ANSWER 7 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 2001:59814 HCAPLUS
DN 134:262576
TI Polyglycosylceramides recognized by **Helicobacter pylori** : analysis by matrix-assisted laser desorption/ionization mass spectrometry after degradation with endo-.beta.-galactosidase and by fast atom bombardment mass spectrometry of permethylated undegraded material
AU Karlsson, Hasse; Larsson, Thomas; **Karlsson, Karl-Anders**; Miller-Podraza, Halina
CS Institute of Medical Biochemistry, Goteborg University, Goteborg, SE 405 30, Swed.
SO Glycobiology (2000), 10(12), 1291-1309
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
CC 6-4 (General Biochemistry)
Section cross-reference(s): 13, 33
AB Human erythrocyte polyglycosylceramides (PGCs) are recognized by the gastric pathogen **Helicobacter pylori** and are based on a successively extended and highly branched **N-acetyllactosamine** core linked to ceramide and substituted by **fucose** and sialic acid. As a step in the identification of the binding epitope, the authors earlier characterized intact PGCs by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry, MALDI-TOF MS. In the present work, PGCs from human blood group O erythrocytes were digested with endo-.beta.-galactosidase (Bacteroides fragilis), an enzyme which cleaves the bond 3Gal.beta.1-4GlcNAc in linear but not branched poly-**N-acetyllactosamine** chains. The enzymic digestion resulted in a mixt. of neutral and sialic acid-contg. glycolipids together with terminal and internal sequences of mainly neutral oligosaccharides. The products were analyzed by MALDI-TOF MS in both pos. and neg. ion mode which gave spectra where the ions could be assigned to structures of the neutral and acidic components, resp. Obsd. were structures which indicated linear extension along both branches. Obsd. at higher masses were fully branched structures obtained by stepwise extension. Most probably further branching may occur along both the (1.fwdarw.3)- and the (1.fwdarw.6)-linked branches to give a partly dendritic structure. Structures with more than one sialic acid substituted could not be obsd. in the MALDI spectrum. Complementary information of the terminal sequences was obtained by FAB-MS anal. of permethylated undegraded PGCs. High-temp. gas chromatog./mass spectrometry of reduced and permethylated products from enzyme hydrolysis documented that Fuc was present in a blood group O sequence, Fuc-Hex-HexN-. **Fucose** may be placed on short (monolactosamine) or longer branches, while sialic acid seems to be restricted to monolactosamine branches. The conclusion is that human erythrocyte PGCs display microheterogeneity within terminal and internal parts of the poly-**N-acetyllactosamine** chains. The first branch from the ceramide end may be located at the second or third Gal and possibly also on the first Gal. Other branches may occur on every **N-acetyllactosamine** unit in fully branched domains, or there may be linear extensions between branches resulting in incompletely branched structures. The extended linear sequences may be present in both 3- and 6-linked antennae. Terminal structures are based on one, two or maybe higher no. of **N-acetyllactosamine** units.
ST blood group O erythrocyte polyglycosylceramide microheterogeneity; **fucose** polyglycosylceramide erythrocyte blood group O; sialic acid polyglycosylceramide erythrocyte blood group O
IT **Blood-group substances**
RL: PRP (Properties)
(O; human blood group O erythrocyte polyglycosylceramides display **fucose** and **sialic** acid microheterogeneity within terminal and internal parts of poly-**N-**

acetyllactosamine chains in relation to recognition by **Helicobacter pylori**)

IT Erythrocyte
Helicobacter pylori
(human blood group O erythrocyte polyglycosylceramides display **fucose** and sialic acid microheterogeneity within terminal and internal parts of poly-**N-acetyllactosamine** chains in relation to recognition by **Helicobacter pylori**)

IT Sialic acids
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(human blood group O erythrocyte polyglycosylceramides display **fucose** and sialic acid microheterogeneity within terminal and internal parts of poly-**N-acetyllactosamine** chains in relation to recognition by **Helicobacter pylori**)

IT Ceramides
RL: PRP (Properties)
(polyglycosylceramides; human blood group O erythrocyte polyglycosylceramides display microheterogeneity within terminal and internal parts of poly-**N-acetyllactosamine** chains in relation to recognition by **Helicobacter pylori**)

IT 2438-80-4, L-**Fucose** **32181-59-2, N-Acetyllactosamine**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(human blood group O erythrocyte polyglycosylceramides display **fucose** and sialic acid microheterogeneity within terminal and internal parts of poly-**N-acetyllactosamine** chains in relation to recognition by **Helicobacter pylori**)

RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

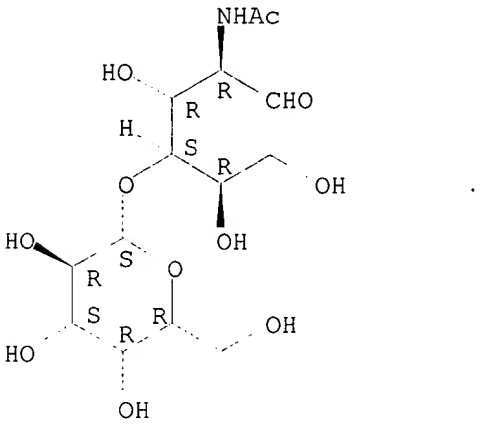
(1) Breimer, M; Advances in Mass Spectrometry 1980, V8, P1097
(2) Ciucanu, I; Carbohydr Res 1984, V131, P209 HCAPLUS
(3) Dabrowski, J; Biochemistry 1988, V27, P5149 HCAPLUS
(4) Dejter-Juszynski, M; Eur J Biochem 1978, V83, P363 HCAPLUS
(5) Domon, B; Glycoconjugate J 1988, V5, P397 HCAPLUS
(6) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(7) Egge, H; J Biol Chem 1985, V260, P4927 HCAPLUS
(8) Johansson, L; Eur J Biochem 1999, V266, P559 HCAPLUS
(9) Karlsson, H; Glycobiology 1999, V9, P765 HCAPLUS
(10) Karlsson, H; Mol Biotech 1994, V1, P165 HCAPLUS
(11) Karlsson, K; Glycobiology, accepted 2000
(12) Karlsson, K; Mol Microbiol 1998, V29, P1 HCAPLUS
(13) Koscielak, J; Eur J Biochem 1976, V71, P9 HCAPLUS
(14) Koscielak, J; Eur J Biochem 1979, V96, P331 HCAPLUS
(15) Levery, S; Biochemistry 1989, V28, P7772 HCAPLUS
(16) Liukkonen, J; J Biol Chem 1992, V267, P21105 HCAPLUS
(17) Loomes, L; Nature 1984, V307, P560 HCAPLUS
(18) Miller-Podraza, H; Biochim Biophys Acta 1993, V1168, P330 HCAPLUS
(19) Miller-Podraza, H; Glycoconjugate J 1996, V13, P453 HCAPLUS
(20) Miller-Podraza, H; Glycoconjugate J 1997, V14, P231 HCAPLUS
(21) Scudder, P; Biochem J 1983, V213, P485 HCAPLUS
(22) Scudder, P; J Biol Chem 1984, V259, P6586 HCAPLUS
(23) Slomiany, B; Eur J Biochem 1980, V113, P27 HCAPLUS
(24) Zdebska, E; Carbohydr Res 1983, V120, P113 HCAPLUS

IT **32181-59-2, N-Acetyllactosamine**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(human blood group O erythrocyte polyglycosylceramides display **fucose** and sialic acid microheterogeneity within terminal and internal parts of poly-**N-acetyllactosamine** chains in relation to recognition by **Helicobacter pylori**)

RN 32181-59-2 HCAPLUS

CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 8 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2001:15310 HCAPLUS
DN 134:204041
TI Salivary agglutinin, which binds Streptococcus mutans and **Helicobacter pylori**, is the lung scavenger receptor cysteine-rich protein gp-340
AU Prakobphol, Akraporn; Xu, Feng; Hoang, Van M.; Larsson, Thomas; Bergstrom, Jorgen; Johansson, Ingegered; Frangsmyr, Lars; Holmskov, Uffe; Leffler, Hakon; Nilsson, Christina; **Boren, Thomas**; Wright, Jo Rae; Stromberg, Nicklas; Fisher, Susan J.
CS Departments of Stomatology, University of California, San Francisco, CA, 94143, USA
SO Journal of Biological Chemistry (2000), 275(51), 39860-39866
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
CC 6-3 (General Biochemistry)
Section cross-reference(s): 13, 14
AB Salivary agglutinin is a high mol. mass component of human saliva that binds Streptococcus mutans, an oral bacterium implicated in dental caries. To study its protein sequence, we isolated the agglutinin from human parotid saliva. After trypsin digestion, a portion was analyzed by matrix-assisted laser/desorption ionization time-of-flight mass spectrometry (MALDI-TOFMS), which gave the mol. mass of 14 unique peptides. The remainder of the digest was subjected to high performance liq. chromatog., and the sepd. peptides were analyzed by MALDI-TOF/post-source decay; the spectra gave the sequences of five peptides. The mol. mass and peptide sequence information showed that salivary agglutinin peptides were identical to sequences in lung (lavage) gp-340, a member of the scavenger receptor cysteine-rich protein family. Immunoblotting with antibodies that specifically recognized either lung gp-340 or the agglutinin confirmed that the salivary agglutining was gp-340. Immunoblotting with an antibody specific to the **sialy Lex** carbohydrate epitope detected expression on the salivary but not the lung glycoprotein, possible evidence of different glycoforms. The salivary agglutinin also interacted with **Helicobacter pylori**, implicated in gastritis and peptic ulcer disease, Streptococcus agalactiae, implicated in neonatal meningitis, and several oral commensal streptococci. These results identify the salivary

agglutinin as gp-340 and suggest it binds bacteria that are important determinants of either the oral ecol. or systemic diseases.

ST saliva agglutinin Streptococcus Helicobacter binding

IT Agglutinins and Lectins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(gp-340; salivary agglutinin, which binds Streptococcus mutans and **Helicobacter pylori**, is the lung scavenger receptor cysteine-rich protein gp-340)

IT **Helicobacter pylori**
Salivary gland
Streptococcus mutans
(salivary agglutinin, which binds Streptococcus mutans and **Helicobacter pylori**, is the lung scavenger receptor cysteine-rich protein gp-340)

RE.CNT 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Aguirre, A; Dysphagia 1989, V4, P95 MEDLINE
(2) Al-Hashimi, I; Arch Oral Biol 1989, V34, P289 HCAPLUS
(3) Azen, E; Adv Hum Genet 1988, V17, P141 HCAPLUS
(4) Bennick, A; Arch Oral Biol 1983, V28, P19 HCAPLUS
(5) Blaser, M; J Clin Invest 1997, V100, P759 HCAPLUS
(6) Bobek, L; J Biol Chem 1993, V268, P20563 HCAPLUS
(7) Boren, T; Science 1993, V262, P1892 HCAPLUS
(8) Brady, L; Infect Immun 1992, V60, P1008 HCAPLUS
(9) Bratt, P; J Dent Res 1999, V78, P1238 HCAPLUS
(10) Carlen, A; Arch Oral Biol 1996, V41, P1133 MEDLINE
(11) Carlen, A; J Dent Res 1995, V74, P1040 HCAPLUS
(12) Carlen, A; J Dent Res 1998, V77, P81 HCAPLUS
(13) Clauser, K; Anal Chem 1999, V71, P2871 HCAPLUS
(14) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(15) Emilson, C; Arch Oral Biol 1989, V34, P335 MEDLINE
(16) Ericson, T; Eur J Biochem 1983, V133, P255 HCAPLUS
(17) Fisher, S; Arch Oral Biol 1987, V32, P509 HCAPLUS
(18) Gans, R; Arch Oral Biol 1990, V35, P487 MEDLINE
(19) Gillece-Castro, B; J Biol Chem 1991, V266, P17358 HCAPLUS
(20) Groenink, J; Antonie Van Leeuwenhoek 1996, V70, P79 HCAPLUS
(21) Hatton, M; Biochem J 1985, V230, P817 HCAPLUS
(22) Holmskov, U; J Biol Chem 1997, V272, P13743 HCAPLUS
(23) Holmskov, U; Proc Natl Acad Sci USA 1999, V96, P10794 HCAPLUS
(24) Ilver, D; Science 1998, V279, P373 HCAPLUS
(25) Kishimoto, E; Infect Immun 1989, V57, P3702 HCAPLUS
(26) Laemmli, U; Nature 1970, V227, P680 HCAPLUS
(27) Lamont, R; Infect Immun 1991, V59, P3446 HCAPLUS
(28) Lenander-Lumikari, M; Caries Res 1992, V26, P371 HCAPLUS
(29) Levine, M; Crit Rev Oral Biol Med 1993, V4, P279 MEDLINE
(30) Levine, M; J Dent Res 1987, V66(suppl), P693
(31) Magnusson, I; Caries Res 1976, V10, P113 MEDLINE
(32) Mandel, I; J Dent Res 1987, V66(suppl), P623
(33) Marshall, B; Lancet 1984, V1, P1311 MEDLINE
(34) Mellersh, A; Br J Vener Dis 1979, V55, P20 MEDLINE
(35) Miyabayashi, H; Helicobacter 2000, V5, P30 MEDLINE
(36) Mollenhauer, J; Nat Genet 1997, V17, P32 HCAPLUS
(37) Murray, P; Infect Immun 1992, V60, P31 HCAPLUS
(38) Nagashunmugam, T; J Infect Dis 1998, V178, P1635 HCAPLUS
(39) Obenauf, S; Infect Immun 1986, V51, P440 HCAPLUS
(40) Oho, T; Infect Immun 1998, V66, P115 HCAPLUS
(41) Prakobphol, A; Biochemistry 1998, V37, P4916 HCAPLUS
(42) Prakobphol, A; Biochemistry 1999, V38, P6817 HCAPLUS
(43) Prakobphol, A; Crit Rev Oral Biol Med 1993, V4, P325 MEDLINE
(44) Rosan, B; Infect Immun 1982, V38, P1056 MEDLINE
(45) Scannapieco, F; J Dent Res 1995, V74, P1360 HCAPLUS
(46) Spahr, H; J Biol Chem 2000, V275, P1351 HCAPLUS

(47) Stenudd, C; Thesis Umea University 1999
(48) Takano, K; Okajimas Folia Anat Jpn 1992, V69, P225 HCAPLUS
(49) Tenovuo, J; J Biol Buccale 1992, V20, P85 MEDLINE
(50) Tino, M; Am J Respir Cell Mol Biol 1999, V20, P759 HCAPLUS
(51) van der Spek, J; Am J Hum Genet 1989, V45, P381 HCAPLUS

L104 ANSWER 9 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:847075 HCAPLUS
DN 134:129472
TI Inhibition of nonopsonic **Helicobacter pylori**-induced activation of human neutrophils by sialylated oligosaccharides
AU **Teneberg, Susann**; Jurstrand, Margaretha; **Karlsson, Karl-Anders**; Danielsson, Dan
CS Institute of Medical Biochemistry, Goteborg University, Goteborg, SE 405 30, Swed.
SO Glycobiology (2000), 10(11), 1171-1181
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
CC 14-3 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 10
AB Certain strains of **Helicobacter pylori** have nonopsonic neutrophil-activating capacity. Some **H.pylori** strains and the neutrophil-activating protein of **H.pylori** (HPNAP) bind selectively to gangliosides of human neutrophils. To det. if there is a relationship between the neutrophil-activating capacity and the ganglioside-binding ability, a no. of **H.pylori** strains, and HPNAP, were incubated with oligosaccharides, and the effects on the oxidative burst of subsequently challenged neutrophils was measured by chemiluminescence and flow cytometry. Both by chemiluminescence and flow cytometry a reduced response was obtained by incubation of **H.pylori** with sialic acid-terminated oligosaccharides, whereas lactose had no effect. The redns. obtained with different sialylated oligosaccharides varied to some extent between the **H.pylori** strains, but in general 3'-sialyllactosamine was the most efficient inhibitor. Challenge of neutrophils with HPNAP gave no response in the chemiluminescence assay, and a delayed moderate response with flow cytometry. Preincubation of the protein with 3'-sialyllactosamine gave a slight redn. of the response, while 3'-sialyllactose had no effect. The current results suggest that the nonopsonic **H.pylori** -induced activation of neutrophils occurs by lectinophagocytosis, the recognition of sialylated glycoconjugates on the neutrophil cell surface by a bacterial adhesin leads to phagocytosis and an oxidative burst with the prodn. of reactive oxygen metabolites.
ST **Helicobacter pylori** neutrophil activation sialylated oligosaccharide
IT **Helicobacter pylori**
(infection; inhibition of nonopsonic **Helicobacter pylori**-induced activation of human neutrophils by sialylated oligosaccharides)
IT Phagocytosis
(inhibition of nonopsonic **Helicobacter pylori** -induced activation of human neutrophils by sialylated oligosaccharides)
IT Carbohydrates, biological studies
Gangliosides
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of nonopsonic **Helicobacter pylori** -induced activation of human neutrophils by sialylated oligosaccharides)
IT Proteins, specific or class

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(neutrophil-activating protein of **H.pylori**;
inhibition of nonopsonic **Helicobacter pylori**
-induced activation of human neutrophils by sialylated
oligosaccharides)

IT 126151-66-4, 3'-Sialyllactosamine
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of nonopsonic **Helicobacter pylori**
-induced activation of human neutrophils by sialylated
oligosaccharides)

IT 63-42-3, Lactose 3001-89-6, 6-Sialyllactose 35890-38-1, 3'-Sialyllactose **98603-84-0** 191667-37-5, 6'-Sialyllactosamine
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of nonopsonic **Helicobacter pylori**
-induced activation of human neutrophils by sialylated
oligosaccharides)

RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Angstrom, J; Glycobiology 1998, V8, P297 HCAPLUS
(2) Ascencio, F; J Med Microbiol 1993, V38, P240 HCAPLUS
(3) Atherton, J; Gastroenterology 1997, V112, P92 MEDLINE
(4) Blaser, M; Cancer Res 1995, V55, P2111 HCAPLUS
(5) Blaser, M; Sci Am 1996, V274, P92
(6) Boren, T; Science 1993, V262, P1892 HCAPLUS
(7) Boyum, A; Tissue Antigens 1974, V4, P269 MEDLINE
(8) Censini, S; Proc Natl Acad Sci USA 1996, V93, P14648 HCAPLUS
(9) Covacci, A; Proc Natl Acad Sci USA 1993, V90, P5791 HCAPLUS
(10) Crabtree, J; Dig Dis Sci 1998, V43(9 Suppl), P40S
(11) Crabtree, J; Gut 1995, V37(Suppl 1), PA3/10
(12) Crabtree, J; J Clin Pathol 1995, V48, P41 MEDLINE
(13) Crabtree, J; Lancet 1991, V338, P332 MEDLINE
(14) Danielsson, D; Dig Dis Sci 1998, V43(Sept 1998 Suppl), P167S
(15) Danielsson, D; J Clin Pathol 2000, V53, P318 MEDLINE
(16) Evans, D; Infect Immun 1988, V56, P2896 HCAPLUS
(17) Evans, D; Infect Immun 1995, V63, P2213 HCAPLUS
(18) Filipe, M; Invest Cell Pathol 1979, V2, P195 HCAPLUS
(19) Fukuda, M; Biochim Biophys Acta 1985, V780, P119 MEDLINE
(20) Fukuda, M; J Biol Chem 1985, V260, P1067 HCAPLUS
(21) Hansen, P; Infect Immun 1999, V67, P3171 HCAPLUS
(22) Ito, Y; J Clin Microbiol 1997, V35, P1710 HCAPLUS
(23) Johansson, L; Glycoconj J 1998, V15, P713 HCAPLUS
(24) Karlsson, K; Methods Enzymol 1987, V138, P212 HCAPLUS
(25) Karlsson, K; Mol Microbiol 1998, V29, P1 HCAPLUS
(26) Kuipers, E; Aliment Pharmacol Ther 1997, V11(Suppl 1), P71
(27) Labigne, A; J Bacteriol 1991, V173, P1920 HCAPLUS
(28) Leyning, H; Mol Microbiol 1992, V6, P2863
(29) Lingwood, C; Infect Immun 1992, V60, P2470 HCAPLUS
(30) Lock, R; APMIS 1988, V96, P299 MEDLINE
(31) Madrid, J; Histochemistry 1990, V95, P179 HCAPLUS
(32) Merritt, J; Biochem J 1993, V289, P919 HCAPLUS
(33) Miller-Podraza, H; Glycoconj J 1996, V13, P453 HCAPLUS
(34) Miller-Podraza, H; Infect Immun 1999, V67, P6309 HCAPLUS
(35) Muthing, J; Glycobiology 1996, V6, P147 MEDLINE
(36) Mysore, J; Gastroenterology 1999, V117, P1316 MEDLINE
(37) Nakamura, S; J Biochem 1991, V115, P1029
(38) Ofek, I; Infect Immun 1988, V56, P539 HCAPLUS
(39) Ohman, L; J Infect Dis 1982, V146, P751 MEDLINE
(40) Pan, Z; J Clin Microbiol 1997, V35, P1344 MEDLINE
(41) Perticarari, S; J Immunol Methods 1994, V170, P117 HCAPLUS
(42) Rautelin, H; Gut 1993, V34, P599 MEDLINE

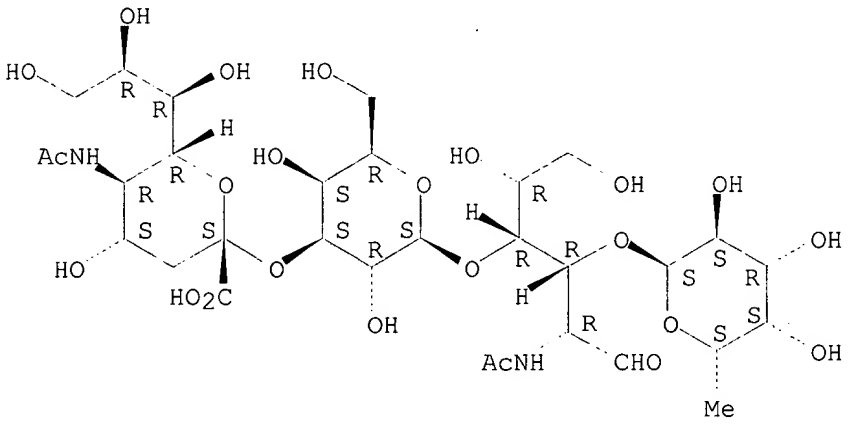
(43) Rautelin, H; Scand J Gastroenterol 1994, V29, P128 MEDLINE
(44) Rautelin, H; Scand J Gastroenterol 1996, V31, P639 MEDLINE
(45) Rest, R; Infect Immun 1985, V50, P116 HCAPLUS
(46) Saitoh, T; FEBS Lett 1991, V282, P385 HCAPLUS
(47) Sharon, N; Lectins 1989, P37
(48) Simon, P; Infect Immun 1997, V65, P750 HCAPLUS
(49) Stroud, M; Biochemistry 1996, V35, P758 HCAPLUS
(50) Stroud, M; Biochemistry 1996, V35, P770 HCAPLUS
(51) Teneberg, S; J Biol Chem 1997, V272, P19067 HCAPLUS
(52) Tonello, F; Mol Microbiol 1999, V34, P238 HCAPLUS
(53) van Doorn, L; J Clin Microbiol 1998, V36, P1271 HCAPLUS
(54) Waldi, D; Dunnschicht-Chromatographie 1962, P496
(55) Xiang, Z; Infect Immun 1995, V63, P94 HCAPLUS
(56) Yoshida, N; Gastroenterology 1993, V105, P1431 MEDLINE

IT **98603-84-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of nonopsonic **Helicobacter pylori** -induced activation of human neutrophils by sialylated oligosaccharides)

RN 98603-84-0 HCAPLUS

CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)]-2-(acetylamino)-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L104 ANSWER 10 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 2000:688099 HCAPLUS

DN 133:276347

TI Use of **fucosylated** sialylated **N-acetyllactosamine** carbohydrate structures for inhibition of bacterial adherence and treatment of conditions related to infection by **Helicobacter pylori** and related gastrointestinal pathogens

IN **Boren, Thomas; Hammarstrom, Lennart; Karlsson, Karl-Anders; Teneberg, Susann**

PA Swed.

SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K031-702
ICS A61K031-715; A61P001-04

CC 1-9 (Pharmacology)

Section cross-reference(s): 63

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000056343	A1	20000928	WO 2000-SE514	20000316 <--
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1169044	A1	20020109	EP 2000-921217	20000316 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002539266	T2	20021119	JP 2000-606247	20000316 <--
PRAI	SE 1999-1007	A	19990319 <--		
	WO 2000-SE514	W	20000316 <--		

AB A **fucosylated** sialylated **N-acetyllactosamine** structure such as a sialyl-Lewis antigen carbohydrate structure, for example **sialyl-Lewis x** and in particular dimeric or repetitive **sialyl-Lewis x**, can be used for the prepn. of a pharmaceutical compn. for the treatment or prophylaxis in humans of conditions involving infection by **Helicobacter pylori** and related pathogens of the human gastrointestinal mucosa. Further, the conditions can be treated through the administration of a **fucosylated** sialylated lactosamine structure, such as a sialyl-Lewis antigen carbohydrate structure, or corresponding antibodies, to patients in need thereof.

ST **fucosylated** sialylated acetyllactosamine carbohydrate Helicobacter therapeutic; Lewis antigen sialyl carbohydrate Helicobacter therapeutic; gastrointestinal pathogen disease **fucosylated** sialylated acetyllactosamine carbohydrate

IT Mutation
(BabA2; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(BabA2; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Le, sialyl; fucosylated sialylated N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Lea; fucosylated sialylated N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(**Leb; fucosylated sialylated N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Lex; fucosylated sialylated N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Stomach, neoplasm**
Stomach, neoplasm
(adenocarcinoma, inhibitors; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Albumins, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(conjugates, with sialylated oligosaccharides; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Antiulcer agents**
(duodenal; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Anti-inflammatory agents
Antiulcer agents
Cell adhesion
Drug delivery systems
Epithelium
Helicobacter pylori
Inflammation
Structure-activity relationship
(**fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Adhesins**
Gangliosides
Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Antitumor agents
(gastric adenocarcinoma; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Stomach, disease**
(gastritis; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Drugs
Pathogen
(gastrointestinal; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Stomach, neoplasm**
Stomach, neoplasm
(lymphoma, inhibitors; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Antibodies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(monoclonal, to (sialyl) Lewis antigens; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Digestive tract**
Stomach
(**mucosa**; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **Digestive tract**
(pathogens; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Antitumor agents
(stomach lymphoma; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Drug delivery systems
(sustained-release; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT Antibodies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(to **fucosylated** sialylated **N-acetyllactosamine** carbohydrate structure; **fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT **32181-59-2D, N-Acetyllactosamine,**
fucosylated and sialylated
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

IT 9003-05-8D, Polyacrylamide, conjugates with sialylated oligosaccharides
21973-23-9 25541-09-7 35890-38-1, 3'-Sialyllactose 35890-39-2, 6'-Sialyllactose 37277-69-3 77538-29-5 77538-32-0 81693-22-3 89678-50-2 91847-18-6 92480-43-8 96119-72-1 101359-93-7 104443-59-6 104443-60-9 104443-62-1 153088-72-3 204118-33-2 242475-89-4
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)

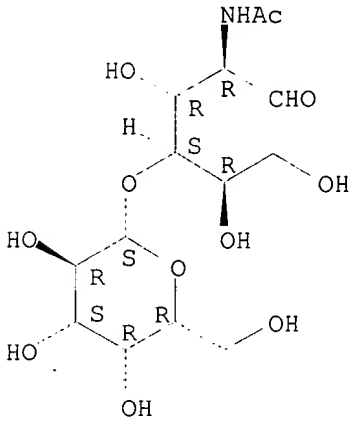
IT 298279-40-0 298279-41-1 298279-42-2 298279-43-3 298279-44-4 298279-45-5
RL: PRP (Properties)
(unclaimed sequence; use of **fucosylated** sialylated **N-acetyllactosamine** carbohydrate structures for inhibition of bacterial adherence and treatment of conditions related to infection by **Helicobacter pylori** and related pathogens)

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Astra Aktiebolag; WO 9500527 A1 1995 HCAPLUS
(2) Hiroyoshi, O; Virchows Arch 1998, V433, P419
(3) Neose Pharmaceuticals Inc; WO 9523605 A1 1995 HCAPLUS
(4) Thomas, B; Science 1993, V262, P1892

IT **32181-59-2D, N-Acetyllactosamine,**
fucosylated and sialylated

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**fucosylated** sialylated **N-acetyllactosamine** carbohydrates for inhibition of bacterial adherence, and therapeutic use)
RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 11 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:768675 HCAPLUS
DN 132:62546
TI **Helicobacter pylori** and neutrophils: sialic acid-dependent binding to various isolated glycoconjugates
AU Miller-Podraza, Halina; Bergstrom, Jorgen; **Teneberg, Susann;** Milh, Maan Abul; Longard, Marianne; Olsson, Britt-Marie; Uggla, Lotta; **Karlsson, Karl-Anders**
CS Institute of Medical Biochemistry, Goteborg University, Goteborg, SE 405 30, Swed.
SO Infection and Immunity (1999), 67(12), 6309-6313
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 14-7 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 10
AB **Helicobacter pylori** has been shown to agglutinate erythrocytes in a sialic acid-dependent manner. However, very few studies have examd. relevant target cells in the human stomach. Neutrophils are required for the onset of gastritis, and the inflammatory reaction may be induced on contact between bacteria and neutrophils. In the present work, glycolipids and glycoproteins were isolated from neutrophils and were studied for binding by overlay with radiolabeled bacteria on thin-layer chromatograms and on membrane blots. There was a complex pattern of binding bands. The only practical binding activity found was sialic acid dependent, since treatment of glycoconjugates with neuraminidase or mild periodate eliminated binding. As shown before for binding to erythrocytes and other glycoconjugates, bacterial cells grown on agar bound to many glycoconjugates, while growth in broth resulted in bacteria that would bind only to polyglycosylceramides, which are highly heterogeneous and branched poly-**N-acetyllactosamine**-contg. glycolipids.
Approx. seven pos. bands were found for glycoproteins, and the traditional ganglioside fraction showed a complex, slow-moving interval with very

strong sialic-acid-dependent binding, probably explained by Fuc substitutions on GlcNAc.

ST Helicobacter binding neutrophil sialate glycoconjugate

IT Neutrophil
(**Helicobacter pylori** sialic acid-dependent binding to glycoconjugates of)

IT **Helicobacter pylori**
(**Helicobacter pylori** sialic acid-dependent binding to glycoconjugates of neutrophil)

IT Gangliosides
Glycosphingolipids
Sialoglycolipids
Sialoglycoproteins
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**Helicobacter pylori** sialic acid-dependent binding to glycoconjugates of neutrophil)

IT Sialic acids
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(conjugates; **Helicobacter pylori** sialic acid-dependent binding to glycoconjugates of neutrophil)

IT Glycoconjugates
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(sialic acid-contg.; **Helicobacter pylori** sialic acid-dependent binding to glycoconjugates of neutrophil)

RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Angstrom, J; Unpublished data
(2) Blaser, M; Gastroenterology 1992, V102, P720 MEDLINE
(3) Boyum, A; Tissue Antigens 1974, V4, P269 MEDLINE
(4) Chmiela, M; FEMS Immunol Med Microbiol 1994, V9, P41 HCAPLUS
(5) Dooley, C; Curr Opin Gastroenterol 1993, V9, P112
(6) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(7) Evans, D; Infect Immun 1988, V56, P2896 HCAPLUS
(8) Evans, D; Infect Immun 1995, V63, P2213 HCAPLUS
(9) Evans, D; J Bacteriol 1993, V175, P674 HCAPLUS
(10) Evans, D; Methods Enzymol 1995, V253, P336 HCAPLUS
(11) Fredlund, H; APMIS 1988, V96, P941 MEDLINE
(12) Graham, D; Gastroenterology 1989, V96, P615 MEDLINE
(13) Hakomori, S; Glycobiology 1998, V8, Pxi HCAPLUS
(14) Harris, P; Gastroenterology 1996, V111, P419 HCAPLUS
(15) Hatz, R; Curr Opin Gastroenterol 1992, V8, P993
(16) Hirmo, S; Glycoconj J 1996, V13, P1005 HCAPLUS
(17) Johansson, L; Anal Biochem 1998, V265, P260 HCAPLUS
(18) Jones, A; J Bacteriol 1997, V179, P5643 HCAPLUS
(19) Karlsson, A; Inflammation 1996, V20, P389 HCAPLUS
(20) Karlsson, H; Glycobiology 1999, V9, P765 HCAPLUS
(21) Karlsson, K; Methods Enzymol 1987, V138, P212 HCAPLUS
(22) Karlsson, K; Methods Enzymol 1987, V138, P220 HCAPLUS
(23) Karlsson, K; Mol Microbiol 1998, V29, P1 HCAPLUS
(24) Kiguchi, K; J Biochem 1990, V107, P8 HCAPLUS
(25) Kist, M; Int J Med Microbiol Virol Parasitol Infect Dis 1993, V280, P58 MEDLINE
(26) Lelwala-Guruge, J; APMIS 1992, V100, P908 MEDLINE
(27) Miller-Podraza, H; Acta Biochim Pol 1998, V45, P439 HCAPLUS
(28) Miller-Podraza, H; Biochim Biophys Acta 1993, V1168, P330 HCAPLUS
(29) Miller-Podraza, H; Glycoconj J 1996, V13, P453 HCAPLUS
(30) Miller-Podraza, H; Glycoconj J 1997, V14, P231 HCAPLUS

(31) Miller-Podraza, H; Glycoconj J 1997, V14, P467 HCAPLUS
(32) Miller-Podraza, H; Infect Immun 1997, V65, P2480 HCAPLUS
(33) Miller-Podraza, H; Unpublished data
(34) Mooney, C; Gut 1991, V32, P853 MEDLINE
(35) Moore, K; J Cell Biol 1992, V118, P445 HCAPLUS
(36) Muthing, J; Carbohydrate Res 1996, V290, P217 MEDLINE
(37) Nedrud, J; Curr Opin Gastroenterol 1997, V13, P71
(38) Nilsson, B; J Biol Chem 1979, V254, P4545 HCAPLUS
(39) O'Toole, P; J Bacteriol 1995, V177, P6049 HCAPLUS
(40) Parkkinen, J; Infect Immun 1986, V54, P37 HCAPLUS
(41) Parsonnet, J; N Engl J Med 1991, V325, P1127 MEDLINE
(42) Parsonnet, J; N Engl J Med 1994, V330, P1267 MEDLINE
(43) Parton, R; Curr Opin Cell Biol 1996, V8, P542 HCAPLUS
(44) Rautelin, H; Gut 1993, V34, P599 MEDLINE
(45) Saitoh, T; FEBS Lett 1991, V282, P385 HCAPLUS
(46) Simon, P; Infect Immun 1997, V65, P750 HCAPLUS
(47) Simons, K; Nature 1997, V387, P569 HCAPLUS
(48) Slomiany, B; Biochem Int 1989, V19, P929 HCAPLUS
(49) Sorice, M; J Lipid Res 1997, V38, P969 HCAPLUS
(50) Spooncer, E; J Biol Chem 1984, V259, P4792 HCAPLUS
(51) Stroud, M; Biochem Biophys Res Commun 1995, V209, P777 HCAPLUS
(52) Stroud, M; Biochemistry 1996, V35, P770 HCAPLUS
(53) Teneberg, S; Unpublished data
(54) Tillack, T; Biochim Bophys Acta 1983, V733, P15 HCAPLUS
(55) Wadstrom, T; J Physiol Pharmacol 1997, V48, P325 HCAPLUS
(56) Warren, J; Lancet 1983, Vi, P1273
(57) Wotherspoon, A; Lancet 1993, V342, P575 MEDLINE
(58) Zhou, B; J Biol Chem 1989, V264, P12272 HCAPLUS

L104 ANSWER 12 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:74012 HCAPLUS
DN 130:278310
TI Glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium
AU Hugosson, Svante; Angstrom, Jonas; Olsson, Britt-Marie; Bergstrom, Jorgen; Fredlund, Hans; Olcen, Per; **Teneberg, Susann**
CS Department of Otorhinolaryngology, Orebro Medical Center Hospital, Orebro, SE 701 85, Swed.
SO Journal of Biochemistry (Tokyo) (1998), 124(6), 1138-1152
CODEN: JOBIAO; ISSN: 0021-924X
PB Japanese Biochemical Society
DT Journal
LA English
CC 6-5 (General Biochemistry)
Section cross-reference(s): 10, 13
AB The glycosphingolipid binding specificities of Haemophilus influenzae and Neisseria meningitidis were investigated as to the binding of radiolabeled bacteria to glycosphingolipids on thin-layer chromatograms. Thereby, similar binding profiles, for the binding of the two bacteria to lactosylceramide, isoglobotriaosylceramide, gangliotriaosylceramide, gangliotetraosylceramide, lactotetraosylceramide, neolactotetraosylceramide, and sialylneolactohexaosylceramide, were obtained. On a closer view the binding preferences of the bacteria could be differentiated into three groups. The first specificity is recognition of lactosylceramide. The second specificity is binding to gangliotriaosylceramide and gangliotetraosylceramide, since conversion of the acetamido group of the N-acetylgalactosamine of gangliotriaosylceramide and gangliotetraosylceramide to an amine prevented the binding of the bacteria, and thus the binding to these two glycosphingolipids represents a sep. specificity from lactosylceramide recognition. Preincubation of H. influenzae with neolactotetraose inhibited the binding to neolactotetraosylceramide, while the binding to

lactosylceramide, gangliotetraosylceramide, or lactotetraosylceramide was unaffected. Thus, the third binding specificity is represented by neolactotetraosylceramide, and involves recognition of other neolacto series glycosphingolipids with linear **N-acetyllactosamine** chains, such as sialyl-neolactohexaosylceramide. The relevance of the detected binding specificities for adhesion to target cells was addressed as to the binding of the bacteria to glycosphingolipids from human granulocytes, epithelial cells of human nasopharyngeal tonsils and human plexus choroideus. Binding-active neolactotetraosylceramide was thereby detected in human granulocytes and the oropharyngeal epithelium.

ST oropharyngeal epithelium glycosphingolipid Neisseria Haemophilus adhesion mol recognition

IT **Cell adhesion**
Haemophilus influenzae
Molecular recognition
Neisseria meningitidis
(glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium)

IT Agglutinins and Lectins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium)

IT Glycosphingolipids
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation); PROC (Process)
(glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium)

IT Epithelium
(oropharyngeal; glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium)

IT 4682-48-8P 11034-93-8P 35960-33-9P 56573-54-7P 60267-39-2P
71012-19-6P 71833-54-0P 71833-57-3P 71833-58-4P 71950-33-9P
71965-57-6P 72067-19-7P 72412-78-3P 72626-26-7P 73201-40-8P
73379-94-9P 73467-80-8P 77538-29-5P 77538-33-1P 79920-77-7P
82030-41-9P 83713-06-8P 84593-23-7P 85305-87-9P 85305-88-0P
86993-34-2P 87501-93-7P 87659-60-7P 88161-63-1P 88844-99-9P
89678-50-2P 91847-18-6P 97666-64-3P 102619-58-9P 104443-59-6P
104443-62-1P 158571-44-9P 186467-26-5P 189201-22-7P 222540-52-5P
222540-53-6P 222540-54-7P 222540-55-8P
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); BIOL (Biological study); PREP (Preparation); PROC (Process)
(glycosphingolipid binding specificities of Neisseria meningitidis and Haemophilus influenzae: detection, isolation, and characterization of a binding-active glycosphingolipid from human oropharyngeal epithelium)

RE.CNT 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Aggarwal, B; Nature 1985, V318, P665 MEDLINE
(2) Andersson, B; J Exp Med 1983, V158, P559 HCAPLUS
(3) Angstrom, J; Glycobiology 1998, V8, P297 HCAPLUS
(4) Baker, N; Infect Immun 1990, V58, P2361 HCAPLUS
(5) Beachey, E; J Infect Dis 1981, V143, P325 HCAPLUS
(6) Bjork, S; J Biol Chem 1987, V262, P6758 MEDLINE
(7) Bock, K; J Biol Chem 1985, V260, P8545 HCAPLUS
(8) Breimer, M; Mass Spectrometry 1980, V8, P1097

(9) Busse, J; J Infect Dis 1996, V175, P77
(10) Dahlberg, T; J Clin Microbiol 1980, V12, P185 MEDLINE
(11) Danielsson, D; Acta Dermatovener 1973, V53, P75 MEDLINE
(12) de Vries, F; Mol Microbiol 1998, V27, P1203 HCAPLUS
(13) Fakih, M; Infect Immun 1997, V65, P1695 HCAPLUS
(14) Falk, K; Arch Biochem Biophys 1979, V192, P164 HCAPLUS
(15) Falk, K; Arch Biochem Biophys 1979, V192, P177 HCAPLUS
(16) Falk, K; Arch Biochem Biophys 1979, V192, P191 HCAPLUS
(17) Firon, N; Infect Immun 1984, V43, P1088 HCAPLUS
(18) Folch, J; J Biol Chem 1957, V226, P497
(19) Geme, J; Am J Respir Crit Care Med 1996, V154, P5192
(20) Gillard, B; Arch Biochem Biophys 1987, V256, P435 HCAPLUS
(21) Gilsdorf, J; Infect Immun 1997, V65, P2997 HCAPLUS
(22) Greenwood, B; Lancet i 1984, P1339 MEDLINE
(23) Hakomori, S; Sphingolipid Biochemistry 1983, V3, P1 HCAPLUS
(24) Handa, S; Jpn J Exp Med 1963, V33, P347 MEDLINE
(25) Hansson, G; Anal Biochem 1985, V146, P158 HCAPLUS
(26) Hansson, G; Biochim Biophys Acta 1983, V750, P214 HCAPLUS
(27) Hartmann, E; Infect Immun 1997, V65, P1729 HCAPLUS
(28) Hugosson, S; Serodiagn Immunother Inf Dis 1996, V8, P213
(29) Iglesias, J; Eur J Biochem 1982, V123, P247 HCAPLUS
(30) Jimenez-Lucho, V; Infect Immun 1990, V58, P2085 HCAPLUS
(31) Kallenius, G; FEMS Microbiol Lett 1980, V7, P297
(32) Karlsson, K; Annu Rev Biochem 1989, V58, P309 HCAPLUS
(33) Karlsson, K; Biochemistry 1974, V13, P3643 HCAPLUS
(34) Karlsson, K; Glycolipid Methodology 1976, P97 HCAPLUS
(35) Karlsson, K; J Biol Chem 1979, V254, P9311 HCAPLUS
(36) Karlsson, K; Methods Enzymol 1987, V138, P212 HCAPLUS
(37) Karlsson, K; Progr Chem Fats Other Lipids 1978, V16, P207 HCAPLUS
(38) Koerner, T; Biochemistry 1983, V22, P2676 HCAPLUS
(39) Koscielak, J; Eur J Biochem 1978, V71, P9
(40) Krivan, H; Proc Natl Acad Sci USA 1988, V85, P6157 HCAPLUS
(41) Larson, G; Carbohydr Res 1987, V161, P281 HCAPLUS
(42) Lee, K; Mol Microbiol 1994, V11, P705 HCAPLUS
(43) Leffler, H; FEMS Microbiol Lett 1980, V8, P127 HCAPLUS
(44) Lingwood, C; Infect Immun 1992, V60, P2470 HCAPLUS
(45) Macher, B; J Biol Chem 1980, V255, P2092 HCAPLUS
(46) Mandrell, R; Infect Immun 1992, V60, P1322 HCAPLUS
(47) Mandrell, R; J Exp Med 1988, V168, P107 HCAPLUS
(48) Mandrell, R; Methods Enzymol 1994, V236, P231 HCAPLUS
(49) Mirelman, D; Microbial Lectins and Agglutinins 1986, P1
(50) Montreuil, J; Biol Cell 1984, V51, P115 HCAPLUS
(51) Noah, N; Bacterial Meningitis 1987, P93
(52) Ofek, I; Bacterial Adhesion to Cells and Tissues 1994, P94
(53) Olcen, P; Acta Pathol Microbiol Scand B 1976, V83, P387
(54) Pesce, M; Clin Chem 1973, V19, P1265 HCAPLUS
(55) Phillips, N; Biochemistry 1992, V31, P4515 HCAPLUS
(56) Phillips, N; Biochemistry 1993, V32, P2003 HCAPLUS
(57) Ritter, G; Arch Biochem Biophys 1987, V257, P370 HCAPLUS
(58) Samuelsson, B; Methods Enzymol 1990, V193, P623 HCAPLUS
(59) Sell, S; Pediatr Infect Dis J 1987, V6, P775 MEDLINE
(60) Sharon, N; Lectins 1989, P37
(61) Sheth, H; Mol Microbiol 1994, V11, P715 HCAPLUS
(62) Smith, A; N Engl J Med 1988, V319, P1012 MEDLINE
(63) Sporsem, O; FEMS Microbiol Lett 1990, V72, P289
(64) Stellner, K; Arch Biochem Biophys 1973, V155, P464 HCAPLUS
(65) Stromberg, N; EMBO J 1990, V9, P2001 MEDLINE
(66) Stroud, M; Biochemistry 1996, V35, P758 HCAPLUS
(67) Stroud, M; Biochemistry 1996, V35, P770 HCAPLUS
(68) Symington, F; J Biol Chem 1987, V262, P11356 HCAPLUS
(69) Taki, T; Lipids 1988, V23, P192 HCAPLUS
(70) Tam, M; Infect Immun 1982, V36, P1042 MEDLINE
(71) Teneberg, S; Glycobiology 1996, V6, P599 HCAPLUS

(72) Teneberg, S; J Biol Chem 1994, V269, P8554 HCAPLUS
(73) Teneberg, S; J Biol Chem 1997, V272, P19067 HCAPLUS
(74) van Alphen, L; FEMS Microbiol Lett 1986, V37, P69
(75) van Alphen, L; Infect Immun 1991, V59, P4473 HCAPLUS
(76) Virji, M; Mol Microbiol 1996, V22, P929 HCAPLUS
(77) Virji, M; Mol Microbiol 1996, V29, P941
(78) Waldi, D; Dunnschicht-Chromatographie 1962, P496
(79) Willoughby, R; J Virol 1990, V64, P4830 HCAPLUS
(80) Yang, H; J Biol Chem 1971, V246, P1192 MEDLINE
(81) Yang, Z; J Biol Chem 1994, V269, P14620 HCAPLUS

L104 ANSWER 13 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:63897 HCAPLUS
DN 128:166039
TI **Helicobacter pylori** adhesin binding **fucosylated** histo-blood group antigens revealed by retagging
AU Ilver, Dag; Arnqvist, Anna; Ogren, Johan; Frick, Inga-Maria; Kersulyte, Dangeruta; Incecik, Engin T.; Berg, Douglas E.; Covacci, Antonello; Engstrand, Lars; **Boren, Thomas**
CS Dep. Microbiol., Umea Univ., Umea, SE-901 87, Swed.
SO Science (Washington, D. C.) (**1998**), 279(5349), 373-377
CODEN: SCIEAS; ISSN: 0036-8075
PB American Association for the Advancement of Science
DT Journal
LA English
CC 15-2 (Immunochemistry)
Section cross-reference(s): 14
AB The bacterium **Helicobacter pylori** is the causative agent for peptic ulcer disease. Bacterial adherence to the human gastric epithelial lining is mediated by the **fucosylated** Lewis b (Leb) histo-blood group antigen. The Leb-binding adhesin, BabA, was purified by receptor activity-directed affinity tagging. The bacterial Leb-binding phenotype was assocd. with the presence of the cag pathogenicity island among clin. isolates of **H. pylori**. A vaccine strategy based on the BabA adhesin might serve as a means to target the virulent type I strains of **H. pylori**.
ST Helicobacter adhesin binding blood antigen Leb; Bab adhesin Helicobacter sequence
IT **Adhesins**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(BabA (blood-group antigen-binding A); **Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)
IT **Adhesins**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(BabB (blood-group antigen-binding B); **Helicobacter pylori** BabA and BabB adhesins in binding **fucosylated** human blood group Leb antigen)
IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(H-1; **Helicobacter pylori** adhesin binding **fucosylated** human blood group **Leb** antigen and)
IT Virulence (microbial)
(**Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)
IT **Cell adhesion**
Helicobacter pylori

Phenotypes
(**Helicobacter pylori** adhesin binding **fucosylated** human blood group Leb antigen)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(**Leb**; **Helicobacter pylori** adhesin binding **fucosylated** human blood group **Leb** antigen)

IT Gene, microbial
RL: PRP (Properties)
(babA1; **Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)

IT Gene, microbial
RL: PRP (Properties)
(babA2; **Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)

IT Gene, microbial
RL: PRP (Properties)
(babB; **Helicobacter pylori** BabA and BabB adhesins in binding **fucosylated** human blood group Leb antigen)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(cagA (cytotoxin-assocd. protein); **Helicobacter pylori** adhesin binding **fucosylated** human blood group Leb antigen in relation to)

IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(cagA; **Helicobacter pylori** adhesin binding **fucosylated** human blood group Leb antigen in relation to)

IT **Stomach**
(**epithelium**; **Helicobacter pylori** adhesin binding **fucosylated** human blood group Leb antigen in)

IT Protein sequences
(of BabA and BabB adhesins of **Helicobacter pylori**)

IT DNA sequences
(of adhesins encoded by BabA1, BabA2, and BabB genes of **Helicobacter pylori**)

IT **Stomach, disease**
(ulcer; **Helicobacter pylori** adhesin binding **fucosylated** human blood group Leb antigen in)

IT 203011-33-0 203011-34-1
RL: PRP (Properties)
(amino acid sequence; **Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)

IT 200890-02-4
RL: PRP (Properties)
(amino acid sequence; **Helicobacter pylori** BabA and BabB adhesins in binding **fucosylated** human blood group Leb antigen)

IT 200889-55-0, GenBank AF001388 202942-15-2
RL: PRP (Properties)
(nucleotide sequence; **Helicobacter pylori** BabA adhesin binding **fucosylated** human blood group Leb antigen)

IT 202636-15-5, GenBank AF001389
RL: PRP (Properties)
(nucleotide sequence; **Helicobacter pylori** BabA and BabB adhesins in binding **fucosylated** human blood group Leb antigen)

L104 ANSWER 14 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 1998:15772 HCAPLUS
DN 128:101086
TI **Helicobacter pylori** blood group antigen-binding adhesin
IN **Boren, Thomas**; Arnqvist, Anna; Normark, Staffan; Ilver, Dag; **Hammarstrom, Lennart**
PA Boren, Thomas, Swed.; Arnqvist, Anna; Normark, Staffan; Ilver, Dag; Hammarstrom, Lennart
SO PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07K014-205
ICS A61K039-106; C07K016-12
CC 15-2 (Immunochemistry)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9747646	A1	19971218	WO 1997-SE1009	19970610 <--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2257826	AA	19971218	CA 1997-2257826	19970610 <--
	AU 9731999	A1	19980107	AU 1997-31999	19970610 <--
	AU 726429	B2	20001109		
	EP 909272	A1	19990421	EP 1997-927563	19970610 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001503606	T2	20010321	JP 1998-501515	19970610 <--
	US 6410719	B1	20020625	US 1998-21560	19980210 <--
PRAI	SE 1996-2287	A	19960610 <--		
	SE 1997-1014	A	19970319 <--		
	US 1997-41040P	P	19970321 <--		
	WO 1997-SE1009	W	19970610 <--		

AB A novel **Helicobacter pylori** blood group antigen binding (BAB) adhesin protein was isolated and purified, whereby said protein or fractions thereof bind specifically to **fucosylated** blood group antigens. The protein sequence of said adhesin is disclosed in this application. Simultaneously the DNA sequences for two genes, babA and babB, producing highly similar proteins, are disclosed. Said adhesin and/or DNA is useful for diagnose and therapy and/or prophylaxis directed against **H. pylori** induced infections, e.g. gastritis and acid peptic disease, i.e. active vaccination. A new Ig compn., which exhibits specific activity to a Lewisb antigen binding **Helicobacter pylori** adhesin, or preferably, monoclonal and/or polyclonal antibodies to said adhesin offer a new and more efficient method of treatment and/or prevention of gastrointestinal diseases, caused by **Helicobacter pylori** or other Helicobacter species, i.e. passive vaccination.

ST **Helicobacter pylori** blood group antigen adhesin; gastric ulcer vaccine **Helicobacter pylori** adhesin

IT Proteins, specific or class
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(73,500 mol. wt.; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT **Adhesins**

RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(BabA (blood-group antigen-binding A); **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT **Adhesins**
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(BabB (blood-group antigen-binding B); **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT Animal cell
Animal tissue
Body fluid
Cattle
Chicken (Gallus domesticus)
Colostrum
Egg yolk
Enterobacteriaceae
Helicobacter
Helicobacter pylori
Lactobacillus
Microorganism
Milk
Staphylococcus
Vaccines
(**Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT Antibodies
Immunoglobulins
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT DNA
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Leb; Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(babA or blood group antigen-binding adhesin; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(babB or blood group antigen-binding adhesin; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**fucosylated; Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive

vaccines)
IT Antibodies
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(monoclonal; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT Antiserums
(monospecific; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT **Digestive tract**
(mucosa; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT DNA sequences
(of blood-group antigen-binding adhesin babA and babB genes of **Helicobacter pylori**)
IT Protein sequences
(of blood-group antigen-binding adhesins BabA and BabB of **Helicobacter pylori**)
IT **Ulcer**
(**peptic**; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT **Stomach, disease**
(ulcer; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT 189032-42-6 200737-81-1
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines)
IT 200890-01-3 200890-02-4
RL: PRP (Properties)
(amino acid sequence; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines in relation to)
IT 200889-55-0 200889-56-1
RL: PRP (Properties)
(nucleotide sequence; **Helicobacter pylori** blood group antigen-binding adhesin and antibody as active and passive vaccines in relation to)

L104 ANSWER 15 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:432048 HCAPLUS
DN 127:146908
TI Avian influenza A viruses differ from human viruses by recognition of sialyloligosaccharides and gangliosides and by a higher conservation of the HA receptor-binding site
AU Matrosovich, M. N.; Gambaryan, A. S.; **Teneberg, S.**; Piskarev, V. E.; Yamnikova, S. S.; Lvov, D. K.; Robertson, J. S.; **Karlsson, K.-A.**
CS M. P. Chumakov Inst. Poliomyelitis Viral Encephalitides, Russian Acad. Med. Sci., Moscow, 142 782, Russia
SO Virology (1997), 233(1), 224-234
CODEN: VIRLAX; ISSN: 0042-6822
PB Academic
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB Avian influenza virus strains representing most hemagglutinin (HA) subtypes were compared with human influenza A (H1N1, H3N2) and B virus isolates, including those with no history of passaging in embryonated hen's eggs, for their ability to bind free N-acetylneuraminic acid (Neu5Ac) and sialyloligosaccharides in a competitive binding assay and to attach to gangliosides in a solid-phase adsorption assay. The avian viruses, irresp. of their HA subtype, showed a higher affinity for sialyl

3-lactose and the other Neu5Ac2-3Gal-terminated oligosaccharides and a lower affinity for sialyl 6-lactose than for free Neu5Ac, indicative of specific interactions between the HA and the 3-linked Gal and poor accommodation of 6-linked Gal in the avian receptor-binding site (RBS). Human H1 and H3 strains, by contrast, were unable to bind to 3-linked Gal, interacting instead with the asialic portion of sialyl-6(**N-acetyllactosamine**). Different parts of this moiety were recognized by H3 and H1 subtype viruses (Gal and GlcNAc, resp.). Comparison of the HA amino acid sequences revealed that residues in positions 138, 190, 194, 225, 226, and 228 are conserved in the avian RBS, while the human HAs harbor substitutions at these positions. A characteristic feature of avian viruses was their binding to Neu5Ac2-3Gal-contg. gangliosides. This property of avian precursor viruses was preserved in early human H3 isolates, but was gradually lost with further circulation of the H3 HA in humans. Consequently, later human H3 isolates, as well as H1 and type B human strains, were unable to bind to short Neu5Ac2-3Gal-terminated gangliosides, an incompatibility that correlated with higher glycosylation of the HA globular head of human viruses. These results suggest that the RBS is highly conserved among HA subtypes of avian influenza virus, while that of human viruses displays distinctive genotypic and phenotypic variability.

ST influenza virus sialyloligosaccharide ganglioside binding hemagglutinin; sialyloligosaccharide binding avian human influenza virus; ganglioside binding avian human influenza virus; hemagglutinin avian human influenza virus

IT **Adhesion, biological**
Influenza A virus
(avian influenza A viruses differ from human viruses by recognition of sialyloligosaccharides and gangliosides and by a higher conservation of the HA receptor-binding site)

IT Hemagglutinins
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(avian influenza A viruses differ from human viruses by recognition of sialyloligosaccharides and gangliosides and by a higher conservation of the HA receptor-binding site)

IT Gangliosides
Sialooligosaccharides
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(avian influenza A viruses differ from human viruses by recognition of sialyloligosaccharides and gangliosides and by a higher conservation of the HA receptor-binding site)

L104 ANSWER 16 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:276640 HCAPLUS
DN 126:341448
TI Screening for the presence of polyglycosylceramides in various tissues: partial characterization of blood group-active complex glycosphingolipids of rabbit and dog small intestines
AU Miller-Podraza, Halina; Stenhagen, Gunnar; Larsson, Thomas; Andersson, Carita; **Karlsson, Karl-Anders**
CS Dep. Med. Biochem., Goteborg Univ., Goteborg, S-413 90, Swed.
SO Glycoconjugate Journal (**1997**), 14(2), 231-239
CODEN: GLJOEW; ISSN: 0282-0080
PB Chapman & Hall
DT Journal
LA English
CC 13-1 (Mammalian Biochemistry)
AB Twenty different human and animal tissues were investigated for the presence of polyglycosylceramides. The glycolipids were isolated by peracetylation of dry tissue residues left after conventional lipid extn., followed by extn. with chloroform and subsequent Sephadex LH-20, Sephadex LH-60 and silica gel chromatog. In most of the cases only trace amts. of

complex glycolipids were found. Distinct bands of glycosphingolipids migrating on TLC plates in a region of brain gangliosides and below were obsd. in bovine erythrocytes, human leukocytes and human colon mucosa. Definite fractions of polyglycosylceramides were isolated from rabbit small intestine, dog small intestine, human placenta and human leukocytes. The polyglycosylceramides of dog and rabbit intestine were characterized by colorimetric anal., methylation anal., mass spectrometry and immunol. assays. The dog material contained branched carbohydrate chains with repeated **fucosylated N-acetyllactosamine** units. Rabbit intestine polyglycosylceramides resembled rabbit erythrocyte polyglycosylceramides with Hex-Hex- terminal determinants but were more complex in respect of sugar compn. and structure. The material isolated from dog intestine showed A, H, Lex and Ley blood group activities. Polyglycosylceramides of human erythrocytes, placenta and leukocytes showed strong binding affinity for **Helicobacter pylori**, while polyglycosylceramide fractions from rabbit and dog intestine were receptor-inactive for this bacterium or displayed only weak and poorly reproducible binding.

ST polyglycosylceramide glycosphingolipid tissue blood group active; dog rabbit intestine glycosphingolipid blood group

IT **Intestine**
(colon, mucosa; screening for presence of polyglycosylceramides in various tissues)

IT Canidae
Helicobacter pylori
Rabbit
(partial characterization of blood group-active complex glycosphingolipids of rabbit and dog small intestines)

IT Blood-group substances
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(partial characterization of blood group-active complex glycosphingolipids of rabbit and dog small intestines)

IT Ceramides
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(polyglycosyl-; screening for presence of polyglycosylceramides in various tissues)

IT Erythrocyte
Leukocyte
Placenta
(screening for presence of polyglycosylceramides in various tissues)

IT Glycolipids
Glycosphingolipids
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(screening for presence of polyglycosylceramides in various tissues)

IT **Intestine**
(small; screening for presence of polyglycosylceramides in various tissues)

L104 ANSWER 17 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:49184 HCAPLUS
DN 126:128068
TI Unexpected carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin. Recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin
AU **karlsson, Karl-Anders; Teneberg, Susann;** Aangstroem, Jonas; Kjellberg, Anders; Hirst, Tomothy R.; Bergstroem, Joergen; Miller-Podraza, Halina
CS Dep. of Medical Biochemistry, Goeteborg Univ., Goeteborg, S-413 90, Swed.
SO Bioorganic & Medicinal Chemistry (1996), 4(11), 1919-1928
CODEN: BMECEP; ISSN: 0968-0896

PB Elsevier
DT Journal
LA English
CC 4-5 (Toxicology)
AB The bacterial protein enterotoxins, cholera toxin (CT) of Vibrio cholerae and heat-labile toxin (LT) of Escherichia coli, induce diarrhea by enhancing the secretory activity of the small intestine of man and rabbit (animal model). This physiol. effect is mediated by toxin binding to a glycolipid receptor, the ganglioside GM1, Gal.beta.3GalNAc.beta.4(NeuAc.alpha.3)Gal.beta.4Glc.beta.1Cer. However, LT, but not CT, was recently shown by us to bind also to paragloboside, Gal.beta.4GlcNAc.beta.3Gal.beta.4Glc.beta.1Cer, identified in the target cells. By mol. modeling of this tetrasaccharide in the known binding site of LT, the saccharide-peptide interaction was shown to be limited to the terminal disaccharide (**N-acetyllactosamine**). This sequence is expressed in many glycoconjugates, and the authors have therefore assayed glycolipids and glycoproteins prepd. from the target tissues. In addn. to paragloboside, receptor activity for LT was detected in glycoproteins of human origin and in polyglycosylceramides of rabbit. However, CT bound only to GM1. Two variants of LT with slightly different sequences, human (hLT) and porcine (pLT), were identical in their binding to target glycoproteins and polyglycosylceramides, but different regarding paragloboside, which was pos. for pLT but neg. for hLT. This difference is discussed on basis of modeling, taking in view the difference at position 13, with Arg in pLT and His in hLT. Although **N-acetyllactosamine** is differently recognized in form of paragloboside by the two toxin variants, we speculate that this sequence in human glycoproteins and rabbit polyglycosylceramides is the basis for the common binding.
ST carbohydrate binding Escherichia heat labile enterotoxin; acetyllactosamine Escherichia heat labile enterotoxin; ganglioside GM1 Escherichia heat labile enterotoxin; paragloboside Escherichia heat labile enterotoxin; cholera toxin binding Escherichia enterotoxin
IT Escherichia coli
(carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)
IT Cerebrosides
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)
IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(cholera; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)
IT Glycoproteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(desialylated; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)
IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(enterotoxin LT; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)
IT Toxins
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(enterotoxins, heat-labile, receptors; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(enterotoxins, heat-labile; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

IT **Intestine**
(small; carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

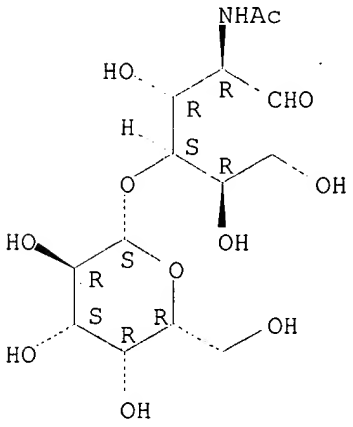
IT 56573-54-7, Paragloboside 71012-19-6, Gangliotetraosylceramide 102619-58-9 104443-62-1, Ganglioside GM1 186467-26-5
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

IT **32181-59-2, N-Acetyllactosamine**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

IT **32181-59-2, N-Acetyllactosamine**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(carbohydrate cross-binding by Escherichia coli heat-labile enterotoxin and recognition of human and rabbit target cell glycoconjugates in comparison with cholera toxin)

RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L104 ANSWER 18 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:9018 HCAPLUS
DN 124:84119
TI **Helicobacter pylori** binds to blood group antigens
AU **Boren, Thomas**; Falk, Per
CS School Medicine, Washington University, St. Louis, USA
SO Scientific American Science & Medicine (**1994**), 1(4), 28-37
CODEN: SASMFP; ISSN: 1068-6746
PB Scientific American, Inc.

DT Journal; General Review
LA English
CC 15-0 (Immunochemistry)
AB A review and discussion with 8 refs. To survive and prosper in the highly acidic human stomach, **H. pylori** produces a potent urease that buffers its immediate environment. To colonize gastric epithelium, the microbe recognizes **fucosylated** blood group antigens known as H and Lewis b expressed on host cell surfaces. Finally, to avoid being flushed away in the rapid turnover of gastric mucosa, **H. pylori** has flagella that make it actively motile. These same characteristics together with secretion of a cytotoxin link **H. pylori** to chronic gastric inflamation and hint at possible ways to clarify its pathogenicity and to devise therapeutic strategies.
ST review Helicobacter blood group antigen binding
IT **Campylobacter pyloridis**
(**Helicobacter pylori** binds to blood group antigens in acid peptic disease.)
IT Blood-group substances
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(H, **Helicobacter pylori** binds to blood group antigens in acid peptic disease.)
IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Leb, Helicobacter pylori** binds to blood group antigens in acid peptic disease.)
IT **Ulcer**
(**peptic, Helicobacter pylori** binds to blood group antigens in acid **peptic** disease.)

L104 ANSWER 19 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:130916 HCAPLUS
DN 122:46463
TI Use of di- or oligosaccharide glycosides as inhibitors of **Helicobacter pylori** adherence
IN Normark, Jan Staffan; Falk, Per; **Boren, Thomas**
PA Swed.
SO PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-70
CC 1-5 (Pharmacology)
Section cross-reference(s): 10, 14, 63
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9418986	A1	19940901	WO 1994-IB23	19940225 <--
	W: AU, BB, BG, BR, BY, CA, CN, CZ, CZ, DE, DE, DK, DK, FI, FI, GE, HU, JP, KG, KP, KR, KZ, LK, LV, MD, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SK, SK, TJ, UA, US, UZ, VN				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2157049	AA	19940901	CA 1994-2157049	19940225 <--
	AU 9460425	A1	19940914	AU 1994-60425	19940225 <--
	EP 690717	A1	19960110	EP 1994-906981	19940225 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	CN 1121311	A	19960424	CN 1994-191793	19940225 <--
	JP 08509467	T2	19961008	JP 1994-518792	19940225 <--
	NO 9503281	A	19950821	NO 1995-3281	19950821 <--
PRAI	DK 1993-222		19930226 <--		

DK 1993-760 19930625 <--
WO 1994-IB23 19940225 <--

AB **H. pylori** has been implicated as a contributing factor in a no. of pathol. conditions, including acute (type B) gastritis, gastric and duodenal ulcers, gastric adenocarcinoma, and gastric lymphoma. The present invention relates to the use of di- or oligosaccharide glycosides contg. at least one terminal L-**fucose** unit for the prepn. of pharmaceutical compns. for the treatment or prophylaxis in humans of conditions involving infection by **H. pylori** in the human gastric mucosa, as well as a method of treating such conditions using such glycosides. Attachment of **H. pylori** to human gastric epithelium using an in situ adherence assay was shown to be inhibited by human colostrum secretory IgA (sIgA), a mol. carrying a highly variable set of N- and O-linked oligosaccharides, while serum IgA was devoid of such inhibitory properties. This inhibitory activity of sIgA could be markedly reduced by .alpha.-L-**fucosidase** treatment of the sIgA. Efforts were made to delineate the **fucosidase** sensitive receptor structure.

ST Helicobacter adherence inhibitor oligosaccharide glycoside; **fucose** glycoside Helicobacter infection stomach inhibition; disaccharide glycoside Helicobacter adherence inhibitor

IT Mucins
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(bovine submaxillary gland; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Campylobacter pyloridis**
(di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Glycoproteins, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Adhesins**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Glycosides
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(disaccharide or oligosaccharide; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Ulcer inhibitors**
(gastric; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Oligosaccharides
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(glycosides; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Colostrum
(human IgA; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Immunoglobulins
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(A, human colostrum; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Immunoglobulins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(A, secretory, human; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**Ley**, tetrasaccharide; **Helicobacter pylori** binding to gastric mucosa inhibition by)

IT **Stomach, neoplasm**
(adenocarcinoma, inhibitors, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric **mucosa**)

IT Adhesion
(bio-, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Stomach, disease**
(chronic gastritis, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric **mucosa**)

IT Oligosaccharides
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(di-, glycosides; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Ulcer inhibitors**
(**duodenal**, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Stomach**
(**epithelium**, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric **mucosa**)

IT **Stomach, neoplasm**
(lymphoma, inhibitors, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric **mucosa**)

IT **Stomach**
(**mucosa**, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric **mucosa**)

IT Neoplasm inhibitors
(stomach adenocarcinoma, di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Neoplasm inhibitors
(stomach lymphoma, di- or oligosaccharide glycosides contg. terminal

fucose as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT **Salivary gland**
(submandibular, bovine mucin of; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT Caseins, biological studies
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(.kappa.-, human; di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT 7578-25-8, Lacto-N-**fucopentaose** I 41263-94-9, 2'-**Fucosyllactose** 41312-47-4, 3-**Fucosyllactose**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**Helicobacter pylori** binding to gastric mucosa inhibition by)

IT 16789-38-1, Lacto-N-**difucohexaose** I
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(Leb; **Helicobacter pylori** binding to gastric mucosa inhibition by)

IT 158753-39-0
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

IT 2438-80-4, L-**Fucose**
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(di- or oligosaccharide glycosides contg. terminal **fucose** as inhibitors of **Helicobacter pylori** adherence to human gastric mucosa)

L104 ANSWER 20 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1994:52202 HCAPLUS
DN 120:52202
TI Attachment of **Helicobacter pylori** to human gastric epithelium mediated by blood group antigens
AU **Boren, Thomas**; Falk, Per; Roth, Kevin A.; Larson, Goran; Normark, Staffan
CS Sch. Med., Washington Univ., St. Louis, MO, 63110, USA
SO Science (Washington, DC, United States) (1993), 262(5141), 1892-5
CODEN: SCIEAS; ISSN: 0036-8075
DT Journal
LA English
CC 15-2 (Immunochemistry)
AB **Helicobacter pylori** is assocd. with development of gastritis, gastric ulcers, and adenocarcinomas in humans. The Lewisb (Leb) blood group antigen mediates **H. pylori** attachment to human gastric mucosa. Sol. glycoproteins presenting the Leb antigen or antibodies to the Leb antigen inhibited bacterial binding. Gastric tissue lacking Leb expression did not bind **H. pylori**. Bacteria did not bind to Leb antigen substituted with a terminal GalNAc.alpha.1-3 residue (blood group A determinant), suggesting that the availability of **H. pylori** receptors might be reduced in individuals of blood group A and B phenotypes, as compared with blood group O individuals.
ST Helicobacter attachment stomach epithelium Lewis antigen

IT **Campylobacter pyloridis**
(attachment of, to human gastric epithelium, Leb blood group antigen in)
IT Blood-group substances
RL: BIOL (Biological study)
(A, **Helicobacter pylori** attachment to human gastric epithelium in relation to)
IT **Blood-group substances**
RL: BIOL (Biological study)
(**Leb**, in **Helicobacter pylori** attachment to human gastric epithelium)
IT Adhesion
(bio-, by **Helicobacter pylori**, to human gastric epithelium, Leb blood group antigen in)
IT **Stomach**
(**epithelium, Helicobacter pylori** attachment to, of humans, Leb blood group antigen in)

L104 ANSWER 21 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1984:528530 HCAPLUS
DN 101:128530
TI Lewis blood group antigens defined by monoclonal anti-colon carcinoma antibodies
AU Blaszczyk, Magdalena; Hansson, Gunnar C.; **Karlsson, Karl Anders**; Larson, Goran; Stromberg, Nicklas; Thurin, Jan; Herlyn, Meenhard; Steplewski, Zenon; Koprowski, Hilary
CS Wistar Inst. Anat. Biol., Philadelphia, PA, 19104, USA
SO Archives of Biochemistry and Biophysics (1984), 233(1), 161-8
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA English
CC 15-2 (Immunochemistry)
AB Monoclonal antibodies directed against humans cancer cells were prepd. by the murine hybridoma technique. These antibodies detect Lewis group antigens as detd. by indirect solid-phase RIA, hapten inhibition studies, and chromatogram binding assay. One monoclonal antibody is specific for the Lea terminal carbohydrate of Gal.beta.1 .fwdarw. 3Glc NAc(4 .rarw. 1.alpha. Fuc).beta.1 .fwdarw. 3LacCeramide. Five monoclonal antibodies react with the Leb terminal carbohydrate sequence of Fuc.alpha.1 .fwdarw. 2Gal.beta.1 .fwdarw. 3GlcNAc(4 .rarw. 1.alpha.Fuc).beta.1 .fwdarw. 3LacCeramide, and 4 of these antibodies are highly specific for this glycolipid and do not react with other similar di- and monofucosylated glycolipids. One of the anti-Leb antibodies cross-reacts with blood group H glycolipid and has binding properties similar to those of the previously described antibody NS-10-17 (Brockhaus, M., et al., 1981). Two antibodies react with both the Lea and Leb antigens, though both bind preferentially to Leb.
ST Lewis blood group antigen carcinoma; monoclonal antibody Lewis antigen carcinoma
IT Glycolipids
Oligosaccharides
RL: BIOL (Biological study)
(monoclonal antibodies reactivity with, Lewis blood group antigens of colon carcinoma of human in relation to)
IT Carcinoma
(monoclonal antibodies to colon, of human, Lewis blood group substances detection by)
IT **Blood-group substances**
RL: PROC (Process)
(**Lewis**, of colon carcinoma, of human, monoclonal antibodies in detection of)
IT **Intestine, neoplasm**
(carcinoma, monoclonal antibodies to, Lewis blood group substances

detection by, of human)
IT Antibodies
RL: BIOL (Biological study)
(monoclonal, in Lewis blood group substances detection, of colon carcinoma)
IT 14116-68-8 21973-23-9 56573-54-7 71950-33-9 73201-40-8
77538-29-5 77538-33-1 78990-73-5 87501-62-0 88161-63-1
91847-17-5 91847-18-6 91847-19-7
RL: BIOL (Biological study)
(monoclonal antibodies reactivity with, Lewis blood group antigens of colon carcinoma of human in relation to)

L104 ANSWER 22 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1982:81680 HCAPLUS
DN 96:81680
TI Lewis blood group fucolipids and their isomers from human and canine intestine
AU McKibbin, John M.; Spencer, William A.; Smith, Edwin L.; Mansson, Jan Eric; **Karlsson, Karl Anders**; Samuelsson, Bo E.; Li, Yu Teh; Li, Su Chen
CS Dep. Biochem., Univ. Alabama, Birmingham, AL, 35294, USA
SO Journal of Biological Chemistry (1982), 257(2), 755-60
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
CC 6-7 (General Biochemistry)
AB Glycolipids contg. linked to N-acetylglucosamine were isolated and characterized from 14 individual human and 13 individual dog intestines, Lewis a isomer fucolipids were isolated, all identical and having the structure Gal(.beta.1.fwdarw.4)[Fuc.alpha.1.fwdarw.3]GlcNAc(.beta.1.fwdarw.3)Gal(.beta.1.fwdarw.4)Glc-ceramide. Lewis b isomer fucolipids were isolated from 12 of the intestines, all identical and having the structure Fuc(.alpha.1.fwdarw.2)Gal(.beta.1.fwdarw.4)[Fuc.alpha.1.fwdarw.3]GlcNAc(.beta.1.fwdarw.3)Gal(.beta.1.fwdarw.4)Glc-ceramide. Lewis a-active glycolipids were isolated as the sole major fucolipid in 6 of the human intestines and differed from the canine isomer only in the position of the linkage of galactose to N-acetylglucosamine, having the .beta.1.fwdarw.3 (type 1) rather than the .beta.1.fwdarw.4 (type 2) linkage. Lewis b-active fucolipids were isolated from 8 human intestines and differed from their canine isomer only in that they, too, had the type 1 rather than the type 2 oligosaccharide chain. Lewis a and b glycolipid isomers commonly co-existed in canine intestine as major fucolipids whereas Lewis a and b glycolipids did not so co-exist in human intestine. In all of the fucolipids, only hydroxylated fatty acids were present and phytosphingosine and sphingosine were the predominant long chain bases.
ST Lewis blood group fucolipid dog intestine
IT Dog
(glycosphingolipids with fucose of intestine of, characterization of, with Lewis blood-group substance activity)
IT **Intestine, composition**
(glycosphingolipids with fucose of, characterization of, from dog and human, with Lewis blood-group substance activity)
IT Glycosphingolipids
RL: BIOL (Biological study)
(with Lewis blood-group activity , fucose-contg., from human and dog intestine)
IT **Blood-group substances**
RL: BIOL (Biological study)
(**Lewis**, glycosphingolipids contg. **fucose** with activity of, characterization of, from human and dog intestine)

L104 ANSWER 23 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1975:527848 HCAPLUS

DN 83:127848
TI Characterization of a human intestinal fucolipid with blood group Lea activity
AU Smith, Edwin L.; McKibbin, John M.; **Karlsson, Karl A.**; Pascher, Irmin; Samuelsson, Bo E.; Li, Yu-Teh; Li, Su-Chen
CS Dep. Biochem., Univ. Alabama, Birmingham, AL, USA
SO Journal of Biological Chemistry (1975), 250(15), 6059-64
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
CC 6-5 (General Biochemistry)
Section cross-reference(s): 15
AB A fucolipid that carried human blood group Lea activity was isolated from human small intestine. It contained fucose, galactose, N-acetylglucosamine, glucose, and ceramide in a M ratio of 1:2:1:1:1. After periodate oxidn. only 1 mol. of galactose and the N-acetylglucosamine remained. Permethylation of the lipid gave derivs. of a terminal fucose and galactose residue together with 2,4,6-tri-O-methylgalactose and 2,3,6-tri-O-methylglucose. After removal of fucose the lipid could be converted to a ceramide trihexoside with .beta.-galactosidase, and this, in turn, to ceramide lactoside by the action of .beta.-N-acetylhexosaminidase. Both enzymes converted the defucosylated deriv. to a ceramide monohexoside. The methylated and the methylated and reduced derivs. of the intact lipid gave ions in mass spectrometry for a terminal hexose and deoxyhexose, a terminal trisaccharide of hexose, deoxyhexose, and N-acetylhexosamine, and terminal tetra- and pentasaccharides. Ceramide fragments characteristic of hydroxy fatty acids with 16,22,23,24 carbons were found together with those of phytosphingosine as the major long chain base. On the basis of these results and the immunologic activity of the fucolipid, a structure is discussed.
ST fucolipid intestine structure; blood group fucolipid intestine
IT **Blood-group substances**
RL: BIOL (Biological study)
(**Lea**, fucolipid of intestine as)
IT **Intestine, composition**
(fucolipid of)
IT Fucolipids
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(of intestine)

=> d all hitstr tot

L109 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:698188 HCAPLUS
DN 130:265794
TI **Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins
AU Ota, Hiroyoshi; Nakayama, J.; Momose, Masanobu; Hayama, Masayoshi; Akamatsu, Taiji; Katsuyama, Tsutomu; Graham, David Y.; Genta, Robert M.
CS Department of Medicine, Veterans Affairs Medical Center, Baylor College of Medicine, Houston, TX, USA
SO **Virchows Archiv (1998), 433**(5), **419**
-426
CODEN: VARCEM; ISSN: 0945-6317
PB Springer-Verlag
DT Journal
LA English
CC 14-7 (Mammalian Pathological Biochemistry)
AB The protective ability of gastric mucins may depend largely on their oligosaccharide chains. We evaluated the effects of **H.**

pylori infection on the glycosylation of gastric mucins. Gastric biopsy specimens from 20 **H. pylori**-infected patients before and after cure of the **H. pylori** infection and 8 normal uninfected volunteers were examd. by immunostaining for simple mucin-type glycoproteins and blood-group-related antigens bearing type 1 chain backbone. The immunoreactivity in different gastric compartments was evaluated. Simple mucin-type glycoproteins and blood-group-related antigens were expressed in surface mucous cells. Simple mucin-type glycoproteins showed antrum-predominant expression in normal volunteers and were found in significantly fewer surface mucous cells in infected patients than in normal volunteers; their expression was restored after eradication of **H. pylori**. **Sialyl Lewisa** and Lewisb were expressed in fewer surface mucous cells after than before eradication. The patterns of glycosylation of gastric mucins vary in different gastric compartments and are reversibly altered by **H. pylori** infection. These alterations may affect the protective functions of gastric mucins.

ST Helicobacter infection mucin glycosylation stomach

IT Glycosylation
Helicobacter pylori
Ulcer
(**Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT Mucins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Lea, sialyl; Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Leb, sialyl; Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT **Stomach**
(antrum; **Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT Infection
(bacterial; **Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

IT **Stomach**
(corpus; **Helicobacter pylori** infection produces reversible glycosylation changes to gastric mucins in humans)

RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Allen, A; Scand J Gastroenterol 1986, V125(Suppl), P71
(2) Boren, T; Science 1993, V262, P1892 HCAPLUS
(3) Byrd, J; Gastroenterology 1997, V113, P455 HCAPLUS
(4) Carneiro, F; Histopathology 1994, V24, P105 MEDLINE
(5) David, L; APMIS Suppl 1992, V27, P162 HCAPLUS
(6) Davidson, J; Gastroenterology 1992, V103, P1552 MEDLINE
(7) Dohi, T; Cancer 1994, V73, P1552 HCAPLUS
(8) Graham, D; Gut 1995, V37, P590
(9) Hirohashi, S; Gann 1984, V75, P540 MEDLINE
(10) Kawano, S; Scand J Gastroenterol 1990, V25, P997 MEDLINE
(11) Kelly, R; J Biol Chem 1995, V270, P4640 HCAPLUS
(12) Kudo, T; J Biol Chem 1996, V271, P9830 HCAPLUS

(13) Kushima, R; Int J Cancer 1993, V55, P904 MEDLINE
(14) Majuri, M; Int J Cancer 1995, V63, P551 HCAPLUS
(15) Markesich, D; Gut 1995, V36, P327 MEDLINE
(16) Murata, K; Am J Clin Pathol 1992, V98, P67 MEDLINE
(17) Nakajima, S; Gastroenterology 1997, V113, P746 MEDLINE
(18) Ohara, S; Comp Biochem Physiol [B] 1993, V106, P153 MEDLINE
(19) Sakamoto, S; Cancer Res 1989, V49, P745 MEDLINE
(20) Sellers, L; Carbohydr Res 1988, V178, P93 HCAPLUS
(21) Shimizu, T; Helicobacter 1996, V1, P197 MEDLINE
(22) Shimizu, T; Helicobacter 1996, V1, P207 MEDLINE
(23) Slomiany, B; Biochem Biophys Res Commun 1987, V142, P783 HCAPLUS
(24) Slomiany, B; J Clin Gastroenterol 1992, V14(Suppl 1), PS114
(25) Strous, G; Crit Rev Biochem Mol Biol 1992, V27, P57 HCAPLUS
(26) Tanegashima, A; Glycoconj J 1996, V13, P537 HCAPLUS
(27) Torrado, J; Am J Clin Pathol 1989, V91, P249 MEDLINE
(28) Torrado, J; Cancer 1990, V66, P1769 MEDLINE
(29) Varki, A; Glycobiology 1993, V3, P97 HCAPLUS
(30) Watkins, W; Glycoproteins 1995, P313 HCAPLUS
(31) Yamaoka, Y; Gastroenterology 1996, V110, P1744 HCAPLUS

L109 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:893094 HCAPLUS
DN 123:276048
TI Oligosaccharides for treating and inhibiting gastric and duodenal ulcers
IN Zopf, David A.; Simon, Paul M.; Roth, Stephen; Mcguire, Edward J.; Langer, Dennis H.
PA Neose Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-715
CC 1-9 (Pharmacology)
Section cross-reference(s): 63
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9523605	A1	19950908	WO 1995-US2388	19950302 <--
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TT, UA				
	RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2183329	AA	19950908	CA 1995-2183329	19950302
	AU 9519323	A1	19950918	AU 1995-19323	19950302
	AU 709149	B2	19990819		
	EP 749314	A1	19961227	EP 1995-911945	19950302
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 09509931	T2	19971007	JP 1995-522955	19950302
	JP 3179108	B2	20010625		
	US 5514660	A	19960507	US 1995-474199	19950607
	US 5753630	A	19980519	US 1996-598431	19960208
	US 5883079	A	19990316	US 1998-75862	19980512
PRAI	US 1994-204515	A	19940302		
	US 1992-922519	B2	19920731		
	US 1993-104483	B1	19930728		
	WO 1995-US2388	W	19950302		
	US 1995-474199	A1	19950607		
	US 1996-598431	A1	19960208		

AB A method for treating and/or inhibiting gastric and duodenal ulcers, comprises administering a pharmaceutical compn. comprising an

oligosaccharide of the following formula: (NeuAc-.alpha.(2-3)-pGal-.beta.(1)-(-X-)m-(-Y-)n-)p-Z; wherein X is a chem. bond or a group capable of linking the p-galactose to either the linking group Y or the multivalent support Z; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C; Y is a linking group; Z is a multivalent support; m is 0 or 1; n is 0 or 1; and p is an integer of 2-1,000. Also described is a pharmaceutical compn. comprising an oligosaccharide of the formula: NeuAc-.alpha.(2-3)-pGal-.beta.(1)-A wherein A is a group capable of bonding to the p-galactose; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C. IC50 value of 3'-sialyl lactose against **Helicobacter pylori** was 6.times.10-3 mmol/mL.
An antiulcer compn. was prepd. by mixing 1g 3'-sialyl lactose and 0.25g ranitidine in water/propylene glycol.

ST ulcer inhibitor oligosaccharide; antiulcer sialyl lactose Helicobacter inhibitor

IT **Campylobacter pyloridis**
(infections; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT **Ulcer inhibitors**
(oligosaccharides for treating and inhibiting gastric and **duodenal** ulcers)

IT Fetuins
Oligosaccharides
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Antibiotics
(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Antihistaminics
(H2, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Leb**, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **Ulcer inhibitors**
(**duodenal**, oligosaccharides for treating and inhibiting gastric and **duodenal** ulcers)

IT Pharmaceutical dosage forms
(oral, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Albumins, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(reaction products, with sialyl lactose; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT 35890-38-1, 3'-Sialyl lactose 35890-38-1D, 3'-Sialyl lactose, reaction products with albumins 35890-39-2
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT 60-54-8, Tetracycline 66357-35-5, Ranitidine 73590-58-6, Omeprazole
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

L109 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:861145 HCAPLUS
DN 123:286509
TI Preparation of fucosylated glycosides as inhibitors of bacterial adherence.
IN Eklind, Karin Ingeborg; Loenn, Hans Roland; Tiden, Anna-Karin Ulla Edit
PA Astra AB, Swed.
SO PCT Int. Appl., 105 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07H015-04
ICS C07H015-08; A61K031-70; A61K047-48
CC 33-3 (Carbohydrates)
Section cross-reference(s): 1
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9500527	A1	19950105	WO 1994-SE604	19940617 <--
	W: AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LU, LV, MD, MG, MN, MW, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2164961	AA	19950105	CA 1994-2164961	19940617
	AU 9470891	A1	19950117	AU 1994-70891	19940617
	EP 706528	A1	19960417	EP 1994-919945	19940617
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
	JP 08512026	T2	19961217	JP 1994-502720	19940617
	LT 3446	B	19951025	LT 1994-1978	19940627
PRAI	DK 1993-761		19930625		
	WO 1994-SE604		19940617		

OS MARPAT 123:286509
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Guanidinyl Y-Z1-R, A-Z2-R, A-Z3-B-Z4-R, A-Z5-B-Z6-C-Z7-R, A-Z8-B-Z9-C-Z10-D-Z11-R, A-Z12-B-Z13-C-Z14-D-Z15-E-Z16-R [Z1-Z16 = O, S, CH2, NR25; R25 = H, alkyl, alkenyl, alkylcarbonyl, (substituted) PhCO; A = Q1; B = Q2; C = Q3; D = Q4; E = Q5; Y = Q6; R = H, alkyl, alkenyl, alkynyl, cycloalkyl, cycloalkylalkyl, alkoxyalkyl, alkylcarbonyl, alkenylcarbonyl, (substituted) cycloalkylalkylcarbonyl, arylcarbonyl, etc.; R1-R3 = H, halo, N3, guanidinyl, alkyl, alkenyl, alkynyl, (substituted) aryl, alkoxyalkyl, etc.; R1A-R4E = R1, YZ1; with provisos], were prepd for therapy or prophylaxis in conditions involving infection by Heliobacter pylori of human gastric mucosa. Thus, Et 3-O-(tri-O-benzyl-.alpha.-L-fucopyranosyl)-4,6-O-benzylidene-2-deoxy-2-phthalimido-1-thio-.beta.-D-glucopyranoside was stirred with N-iodosuccinimide, mol. sieves, and CF3CO2H in CH2Cl2/Et2O to give 97% Me 4,6-O-benzylidene-3-O-(tri-O-benzyl-.alpha.-fucopyranosyl)-2-deoxy-2-phthalimido-.beta.-D-glucopyranoside. This was refluxed 20 h with N2H4 in aq. EtOH followed by acetylation of the crude product to give Me 2-acetamido-3-O-(2,3,4-tri-O-benzyl-.alpha.-L-fucopyranosyl)-4,6-O-benzylidene-2-deoxy-.beta.-D-glucopyranoside. The latter was hydrogenolyzed at 200 kPa over Pd/C in AcOH/EtOAc/H2O to give 90% Me 2-acetamido-2-deoxy-3-O-.alpha.-L-fucopyranosyl-D-glucopyranoside. Title compds. gave 34-93% inhibition of

binding of **Helicobacter pylori** to human gastric tissue. Use of title compds. with various antibiotics, antacids, gastric secretion inhibitors, antigastritis drugs, and antiulcer drugs, is claimed.

ST fucosylated glycoside prepn bacterial adherence inhibitor; **helicobacter pylori** adhesion inhibitor fucosylated glycoside; gastric mucosa **helicobacter pylori** adhesion inhibitor

IT **Ulcer inhibitors**
(fucosylated glycosides as inhibitors of **Helicobacter pylori** adherence to gastric mucosa)

IT **Campylobacter pyloridis**
(prepn. of fucosylated glycosides as inhibitors of **Helicobacter pylori** adherence to gastric mucosa)

IT 97242-89-2P 125739-61-9DP, polyacrylamide conjugate 169151-24-0P 169151-25-1P 169151-26-2DP, bovine serum albumin conjugate 169151-27-3P 169151-28-4P 169151-29-5DP, human serum albumin conjugate 169151-30-8DP, human serum albumin conjugate 169151-31-9DP, polyacrylamide conjugate 169151-32-0DP, polyacrylamide conjugate 169151-33-1DP, polyacrylamide conjugate 169151-63-7DP, polyacrylamide conjugate
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of fucosylated glycosides as inhibitors of bacterial adherence)

IT 79-06-1, Acrylamide, reactions 463-71-8, Thiophosgene 624-95-3 814-68-6, Acryloyl chloride 1517-05-1, 2-Azidoethanol 3068-32-4, Acetobromogalactose 6338-55-2 99409-26-4 99409-32-2 99409-33-3 99409-34-4 110089-18-4 117252-99-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(prepn. of fucosylated glycosides as inhibitors of bacterial adherence)

IT 125739-61-9P 130539-43-4P 131545-03-4P 131545-04-5P 131566-40-0P 132932-06-0P 162466-43-5P 169151-31-9P 169151-32-0P 169151-34-2P 169151-35-3P 169151-36-4P 169151-37-5P 169151-38-6P 169151-40-0P 169151-41-1P 169151-42-2P 169151-43-3P 169151-44-4P 169151-45-5P 169151-46-6P 169151-47-7P 169151-48-8P 169151-49-9P 169151-50-2P 169151-51-3P 169151-52-4P 169151-53-5P 169151-54-6P 169151-55-7P 169151-56-8P 169151-57-9P 169151-58-0P 169151-59-1P 169151-60-4P 169151-61-5P 169151-62-6P 169151-63-7P 169151-64-8P 169151-65-9P 169151-66-0P 169273-06-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of fucosylated glycosides as inhibitors of bacterial adherence)

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L117 ANSWER 1 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2001:472908 HCAPLUS
DN 135:72142
TI Modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-containing sugar biosynthesis
IN Endo, Tetsuo; Koizumi, Satoshi; Tabata, Kazuhiko; Ozaki, Akio
PA Kyowa Hakko Kogyo Co., Ltd., Japan
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
IC ICM C12N015-09
ICS C12N001-21; C12N009-10; C12P019-18; C12N001-21; C12R001-19
CC 3-2 (Biochemical Genetics)
Section cross-reference(s): 7, 10

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001046400	A1	20010628	WO 2000-JP9033	20001220 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2001022216	A5	20010703	AU 2001-22216	20001220 <--
	EP 1243647	A1	20020925	EP 2000-985799	20001220 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
PRAI	JP 1999-362243	A	19991221 <--		
	WO 2000-JP9033	W	20001220		

AB Recombinant DNA coding for **Helicobacter pylori** .alpha.1,2-**fucosyltransferase** (FucT) with modification in poly(C) sequence, TAA repeats, or AAAAAAG sequences, and designed with preferred codon usage, and use in biosynthesis of **fucose**-contg. oligosaccharides, are disclosed. A **fucose**-contg. sugar can be economically produced in a large amt. by bringing a acceptor sugar into contact with a microorganism capable of producing GTP from a GTP precursor and a microorganism capable of producing GDP-**fucose** from a sugar and GTP in an aq. medium. The acceptor sugar is an oligosaccharide contg. galactose at the non-reducing end. The oligosaccharide moiety is either lactose, **N-acetyl lactosamine**, Lewis X, or Lewis a. A **fucose**-contg. sugar such as **fucosyl** lactose, **fucosyl N-acetyl lactosamine**, Lewis Y, or Lewis b are produced. GTP precursors such as guanine, xanthine, hypoxanthine, guanosine, xanthosine, inosine, guanosine-5'-monophosphate, xanthosine-5'-monophosphate, or inosine-5'-monophosphate, can be used. Glucose, fructose, or mannose can be used for GDP-**fucose** prodn. Corynebacteria such as Corynebacterium ammoniagenes can be used. Microorganism having elevated activity of glucokinase (glk gene), phosphomannomutase (manB gene), mannose-1-phosphate guaniryltransferase (manC gene), phosphoglucomutase (pgm gene), phosphofructokinase (pfk gene), GDP-mannose 4,6-dehydratase (gmd gene), or GKDM epimerase/reductase (wcaG gene), can be used. **Helicobacter pylori** lipopolysaccharides (LPS) contain complex carbohydrates known as Lewis antigens which may contribute to the pathogenesis and adaptation of the bacterium. Involved in the biosynthesis of Lewis antigens is an .alpha.1,2-**fucosyltransferase** (FucT) that adds **fucose** to the terminal .beta.Gal unit of the O-chain of LPS. Recently, the **H. pylori** (Hp) .alpha.1,2-FucT-encoding gene (fucT2) was cloned and analyzed in detail. In contrast to the normal mammalian .alpha.1,2-FucT (H or Se enzyme), Hp .alpha.1,2-FucT prefers to use Lewis X [.beta.Gal1-4(.alpha.Fuc1-3).beta.GlcNAc] rather than LacNAc [.beta.Gal1-4.beta.GlcNAc] as a substrate, suggesting that **H. pylori** uses a novel pathway (via Lewis X) to synthesize Lewis Y. Hp .alpha.1,2-FucT also acts on type 1 acceptor [.beta.Gal1-3.beta.GlcNAc] and Lewis a [.beta.Gal1-3(.alpha.Fuc1-4).beta.GlcNAc], which provides **H. pylori** with the potential to synthesize H type 1 and Lewis b epitopes. The ability to transfer **fucose** to a **monofucosylated** substrate (Lewis X or Lewis a) makes Hp .alpha.1,2-FucT distinct from normal mammalian .alpha.1,2-FucT.

ST Helicobacter **fucosyltransferase** oligosaccharide lewis antigen biosynthesis

IT Genetic element

RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(AAAAAAG; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT **Blood-group substances**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation)
(**Le**, Y; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Lea**, oligosaccharide moiety of acceptor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT **Blood-group substances**
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation)
(**Leb**; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Lex**, oligosaccharide moiety of acceptor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Galactooligosaccharides
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**fucose** acceptor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Oligosaccharides, biological studies
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation)
(**fucose**-contg.; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Codon usage
DNA sequences
Helicobacter pylori
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process); USES (Uses)
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Genetic element
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(poly(C); modified **Helicobacter pylori** .alpha.-1,2-

fucosyltransferase gene and use in **fucose**-contg. sugar biosynthesis)

IT Escherichia coli
(recombinant expression in; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Repetitive DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(tandem, TAA; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT Corynebacterium
Corynebacterium ammoniagenes
(use in **fucose**-contg. sugar biosynthesis; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 58-63-9, Inosine 68-94-0, Hypoxanthine 69-89-6, Xanthine 73-40-5, Guanine 85-32-5, Guanosine-5'-monophosphate 118-00-3, Guanosine, biological studies 131-99-7, Inosine-5'-monophosphate 146-80-5, Xanthosine 523-98-8, Xanthosine-5'-monophosphate
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(GTP precursor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 9001-36-9P, Glucokinase 9001-80-3P, Phosphofructokinase 9001-81-4P, Phosphoglucomutase 37211-59-9P, GDP-mannose 4,6-dehydratase 37278-24-3P, Mannose-1-phosphate guanylyltransferase 59536-73-1P, Phosphomannomutase 113756-18-6P, GDP-4-keto-6-deoxymannose 3,5-epimerase 4-reductase
RL: BPN (Biosynthetic preparation); CAT (Catalyst use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(elevated activity of, use in **fucose**-contg. sugar biosynthesis; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); BIOL (Biological study); PREP (Preparation); USES (Uses)
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 60797-31-1P 108795-32-0P, **Fucosyl** lactose
RL: BPN (Biosynthetic preparation); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PREP (Preparation)
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 86-01-1D, GTP, precursor 15839-70-0, GDP-**fucose**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 86-01-1, 5'-GTP
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)

(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 347429-52-1
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 63-42-3, Lactose **32181-59-2, N-Acetyl lactosamine**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(oligosaccharide moiety of acceptor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 339966-80-2, 6: PN: WO0177313 SEQID: 6 unclaimed DNA 339966-81-3, 7: PN: WO0177313 SEQID: 7 unclaimed DNA 339966-82-4, 8: PN: WO0177313 SEQID: 8 unclaimed DNA 339966-83-5, 9: PN: WO0177313 SEQID: 9 unclaimed DNA 339966-84-6 339966-85-7 339966-86-8 347435-33-0, 3: PN: WO0146400 SEQID: 3 unclaimed DNA 347435-34-1, 4: PN: WO0146400 SEQID: 4 unclaimed DNA 347435-35-2, 5: PN: WO0146400 SEQID: 5 unclaimed DNA 347435-36-3, 6: PN: WO0146400 SEQID: 6 unclaimed DNA 347435-37-4, 7: PN: WO0146400 SEQID: 7 unclaimed DNA 347435-38-5, 8: PN: WO0146400 SEQID: 8 unclaimed DNA 347435-39-6, 9: PN: WO0146400 SEQID: 9 unclaimed DNA 347435-40-9 347435-41-0 347435-42-1 347435-43-2 347435-44-3 347435-45-4 347435-46-5 347435-47-6 347435-48-7 347435-49-8 347435-57-8
RL: PRP (Properties)
(unclaimed nucleotide sequence; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 224432-11-5
RL: PRP (Properties)
(unclaimed protein sequence; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

IT 50-99-7, D-Glucose, biological studies 57-48-7, D-Fructose, biological studies 3458-28-4, D-Mannose
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(use for GDP-**fucose** prodn.; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

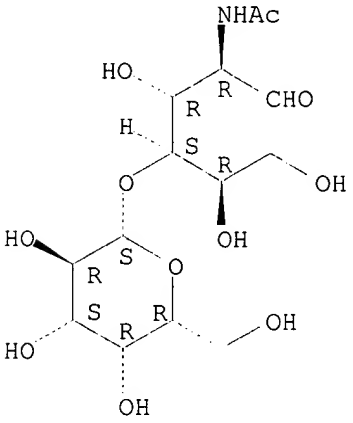
RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Ge, W; Molecular Microbiology 1999, V31(4), P1265
(2) Governors Of The University Of Alberta; AU 1022500 A
(3) Governors Of The University Of Alberta; WO 0026383 A1 2000 HCAPLUS
(4) Kyowa Hakko Kogyo Co Ltd; CA 2237849 A HCAPLUS
(5) Kyowa Hakko Kogyo Co Ltd; AU 4220397 A
(6) Kyowa Hakko Kogyo Co Ltd; EP 870841 A1 HCAPLUS
(7) Kyowa Hakko Kogyo Co Ltd; WO 9812343 A1 1998 HCAPLUS

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); BIOL (Biological study); PREP (Preparation); USES (Uses)
(modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)

RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **32181-59-2, N-Acetyl lactosamine**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(oligosaccharide moiety of acceptor; modified **Helicobacter pylori** .alpha.-1,2-**fucosyltransferase** gene and use in **fucose**-contg. sugar biosynthesis)
RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L117 ANSWER 2 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2001:208394 HCAPLUS
DN 134:247231
TI Transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection
IN Paton, Adrienne; Morona, Renato; Paton, James
PA Women's and Children's Hospital, Australia; Luminis Pty Ltd.
SO PCT Int. Appl., 94 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N001-21
ICS A61K031-7028; A61K031-702; A61K035-74
CC 1-5 (Pharmacology)
Section cross-reference(s): 3, 10
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001019960	A1	20010322	WO 2000-IB1349	20000909 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1214396	A1	20020619	EP 2000-958947	20000909 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
	BR 2000013915	A	20021119	BR 2000-13915	20000909 <--
PRAI	AU 1999-2757	A	19990910 <--		

WO 2000-IB1349 W 20000909

AB Transgenic microorganisms that carry mimics of the endogenous carbohydrate ligand for a bacterial toxin or virulence factor are described for use in the control of infection or intoxication. These microorganisms can be used as a means to competitively inhibit the binding of toxins or adhesins to receptors of mucosal surfaces, esp. gastrointestinal surface. In particular chimeric sugar moieties have been made for lipopolysaccharides, in recombinant microorganism that present multiple copies of the oligosaccharides. The oligosaccharide moieties so presented act as receptor mimic for toxins and adhesins. A no. have been synthesized and have been shown to confer protection against attack by pathogenic organisms or their products in vitro and an in vivo.

ST adhesin carbohydrate ligand mimic infection inhibition; Shiga toxin carbohydrate ligand mimic infection inhibition; transgenic bacteria carbohydrate ligand mimic infection inhibition

IT **Blood-group substances**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Lea, sialyl**, mimics of, in control of bacterial infection; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT **Blood-group substances**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**Lex, sialyl**, mimics of, in control of bacterial infection; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(Shiga, mimics of cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Acanthamoeba
Candida albicans
Chlamydia trachomatis
Entamoeba histolytica
Haemophilus influenzae
Haemophilus parainfluenzae
Helicobacter pylori
Pseudomonas
Streptococcus pneumoniae
(adhesin ligand mimics for control of infection by; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Campylobacter jejuni
(as decoy for heat-labile enterotoxin; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Oligosaccharides, biological studies
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(as ligands for pathogenic bacteria; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Antibacterial agents
(bacteria presenting carbohydrate ligands for virulence factors as; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Nucleotides, biological studies

RL: BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
(biosynthesis of, in manuf. of mimic ligands for virulence factors; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Glycolipids
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(carbohydrate moieties of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Adhesins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(cholera, mimics of cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Inflammation
(control of bacterial binding to cell surfaces in treatment of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Cat (Felis catus)
Cattle
Chicken (Gallus domesticus)
Dog (Canis familiaris)
Duck
Goat
Goose
Horse (Equus caballus)
Rabbit
Sheep
Swine
Turkey
(control of bacterial infection of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Clostridium difficile
(control of infection by; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Digestive tract
(delivery of bacteria presenting mimic ligands for virulence factors to; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Escherichia coli
(enterotoxigenic, control of infection by; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL

(Biological study); PROC (Process); USES (Uses)
(enterotoxins, Clostridium, mimics of cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(enterotoxins, heat-labile, cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Toxins
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(enterotoxins, mimics of cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Polysaccharides, biological studies
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(exopolysaccharides, expression hosts deficient in; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Bifidobacterium
Escherichia coli
Intestinal bacteria
Lactobacillus
Lactococcus
Salmonella enterica typhimurium
Salmonella typhimurium
(expression host; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Capsule (microbial)
(expression hosts deficient in; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection).

IT Environmental analysis
(for bacterial toxins; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(lgtA, expression in Escherichia coli of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(lgtB, expression in Escherichia coli of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(lgtC, expression in Escherichia coli of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on

their surfaces and their use in controlling infection)

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(lgtD, expression in Escherichia coli of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Gene, microbial
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(lgtE, expression in Escherichia coli of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Aeromonas
Campylobacter
Citrobacter
Clostridium
Entamoeba
Escherichia
Haemophilus
Helicobacter
Klebsiella
Neisseria
Pasteurella
Rotavirus
Salmonella
Shigella
Staphylococcus
Streptococcus
Vibrio
Yersinia
(ligands for virulence factors of; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Pilus
(mimics of carbohydrates ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Sialic acids
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oligosaccharides, mimics for virulence factor ligands contg.; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT Drug delivery systems
(oral, bacteria presenting carbohydrate ligands for virulence factors in; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 131-48-6, N-Acetylneuraminic acid 499-40-1 1811-31-0, N-Acetylgalactosamine 3371-50-4 13117-26-5 24656-24-4 29923-15-7 41744-59-6 **54827-14-4D**, GM3, NeuNAc and NeuGc derivs. 330624-92-5
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(as inhibitor of bacterial binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 330624-91-4
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study);

FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(as inhibitor of bacterial binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding rides on their surfaces and their use in controlling infection)

IT 133-89-1, UDP glucose 528-04-1 2956-16-3, UDP galactose 3063-71-6 3123-67-9, GDP mannose 3616-06-6, UDP xylose 15839-70-0, GDP **fucose**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(as substrate for biosynthesis of carbohydrate ligand mimics; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 11000-04-7, Colicin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(expression hosts resistant to; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT **52725-57-2**, Gb3 synthase 321976-25-4, Sialyltransferase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gene for, expression in transgenic Escherichia coli; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 9033-07-2, Glycosyltransferase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(in manuf. of mimic ligands for virulence factors; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT **11034-93-8**, Globotetraosyl ceramide **71965-57-6**, Globotriosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(inhibition of Shiga toxin binding to animal cells via; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT **37758-47-7**, GM1 **71012-19-6**, Asialo-GM1
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(inhibition of heat labile enterotoxin binding to animal cells via; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 13007-32-4, Lacto-N-neotetraose 77356-46-8
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(manuf. of, as inhibitor of Clostridium binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT **32181-59-2** 66580-68-5 75660-79-6, Globotetraose
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(manuf. of, as inhibitor of Shiga toxin binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 59-23-4D, D-Galactose, oligosaccharides, biological studies 2438-80-4D, L-**Fucose**, oligosaccharides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mimics for virulence factor ligands contg.; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 107231-12-9, Botulin
RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(mimics of cell surface ligands for; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

IT 331413-54-8 331477-30-6 331477-31-7 331477-32-8 331477-33-9
331477-34-0 331477-35-1 331477-36-2 331477-37-3 331477-38-4
331477-39-5 331477-40-8 331477-41-9 331477-42-0 331477-43-1
331477-44-2 331477-45-3 331477-46-4 331477-47-5 331477-48-6
331477-49-7 331477-50-0 331477-51-1 331477-52-2 331477-53-3
RL: PRP (Properties)
(unclaimed sequence; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Donnelly, J; Nature Medicine 2000, V6(3), P257 HCAPLUS
(2) Johnson, K; Glycoconjugate Journal 1999, V16, P141 HCAPLUS
(3) Krivan, H; US 5696000 1997 HCAPLUS
(4) Paton, A; Nature Medicine 2000, V6(3), P265 HCAPLUS
(5) Phillips, N; J Biol Chem USA 2000, V275(7), P4747 HCAPLUS
(6) Rafter, D; US 5849714 1998 HCAPLUS

IT **54827-14-4D**, GM3, NeuNAc and NeuGc derivs.
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(as inhibitor of bacterial binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

RN 54827-14-4 HCAPLUS
CN Ganglioside GM3 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **52725-57-2**, Gb3 synthase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gene for, expression in transgenic Escherichia coli; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

RN 52725-57-2 HCAPLUS
CN Galactosyltransferase, uridine diphosphogalactose-lactosylceramide (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **11034-93-8**, Globotetraosyl ceramide **71965-57-6**, Globotriosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(inhibition of Shiga toxin binding to animal cells via; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)

RN 11034-93-8 HCAPLUS

CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-.alpha.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

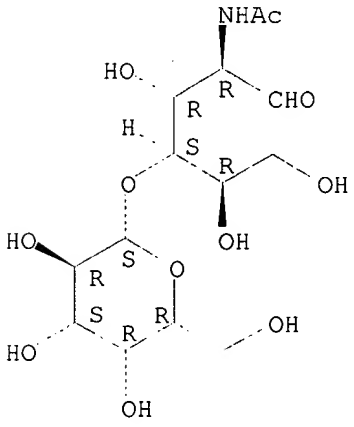
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71965-57-6 HCAPLUS
CN Ceramide, 1-O-(O-.alpha.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl)- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **37758-47-7**, GM1 **71012-19-6**, Asialo-GM1
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(inhibition of heat labile enterotoxin binding to animal cells via; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)
RN 37758-47-7 HCAPLUS
CN Ganglioside GM1 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **32181-59-2**
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); USES (Uses)
(manuf. of, as inhibitor of Shiga toxin binding to animal cells; transgenic microorganisms presenting mimics of mammalian adhesin-binding oligosaccharides on their surfaces and their use in controlling infection)
RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L117 ANSWER 3 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 2001:59943 HCAPLUS
DN 134:236268
TI Large-scale production of GDP-**fucose** and Lewis X by bacterial coupling
AU Koizumi, S.; Endo, T.; Tabata, K.; Nagano, H.; Ohnishi, J.; Ozaki, A.
CS Tokyo Research Laboratories, Kyowa Hakko Kogyo Co., Ltd., Tokyo, 194-8533, Japan
SO Journal of Industrial Microbiology & Biotechnology (**2000**), 25(4), 213-217
CODEN: JIMBFL; ISSN: 1367-5435
PB Nature Publishing Group
DT Journal
LA English
CC 16-2 (Fermentation and Bioindustrial Chemistry)
AB A large-scale prodn. system of GDP-**fucose** (GDP-Fuc) and **fucosylated** oligosaccharides was established by the combination of recombinant Escherichia coli cells overexpressing GDP-Fuc biosynthetic genes and Corynebacterium ammoniagenes cells. E. coli cells overexpressed the genes for glucokinase, phosphomannomutase, mannose-1-phosphate guanylyltransferase, GDP-mannose (GDP-Man) dehydratase, and GDP-4-keto-6-deoxy-mannose (GKDM) epimerase/reductase as well as phosphoglucomutase and phosphofructokinase. C. ammoniagenes contributed to the formation of GTP from GMP. GDP-Fuc accumulated to 29 mM (18.4 g l-1) after a 22-h reaction starting with GMP and mannose through introducing the two-step reaction to overcome the inhibition of GDP-Fuc on GDP-Man dehydratase activity. When E. coli cells overexpressing the .alpha.1,3-**fucosyltransferase** gene of **Helicobacter pylori** were put into the GDP-Fuc prodn. system, Lewis X [Gal.beta.1-4(Fuc.alpha.1-3)GlcNAc] was produced at an amt. of 40 mM (21 g l-1) for 30 h from GMP, mannose, and **N-acetyllactosamine**. The prodn. system through bacterial coupling can be applied to the industrial manuf. of **fucosylated** oligosaccharides.
ST GDP **fucose** Lewis X antigen manuf bacteria coupling; **fucosylated** oligosaccharide prodn bacteria coupling
IT **Blood-group substances**
RL: BMF (Bioindustrial manufacture); BIOL (Biological study); PREP (Preparation)
(**Lex**; large-scale prodn. of GDP-**fucose** and **Lewis** X by bacterial coupling)
IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(fucT; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)
IT Oligosaccharides, preparation
RL: BMF (Bioindustrial manufacture); BIOL (Biological study); PREP (Preparation)
(**fucose**-contg.; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)
IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(glk; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)
IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(gmd; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)
IT Corynebacterium ammoniagenes

Fermentation
Genetic engineering
Helicobacter pylori
Molecular cloning
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(manB; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(manC; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(pfkB; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(pgm; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Escherichia coli
(recombinant; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT Gene, microbial
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(wcaG; large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT 9001-36-9P, Glucokinase 9001-80-3P, Phosphofructokinase. 9001-81-4P, Phosphoglucomutase 37211-59-9P, GDP-mannose dehydratase 37278-24-3P, Mannose-1-phosphate guanylyltransferase 59536-73-1P, Phosphomannomutase 68247-53-0P, .alpha.1,3-**Fucosyltransferase** 113756-18-6P, GDP-4-keto-6-deoxy-mannose epimerase/reductase
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT 15839-70-0P, GDP-**fucose**
RL: BMF (Bioindustrial manufacture); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT 86-01-1P, 5' GTP
RL: BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation); PROC (Process)
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

IT 85-32-5, 5' GMP 3458-28-4, D Mannose **32181-59-2, N-Acetyl lactosamine**

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

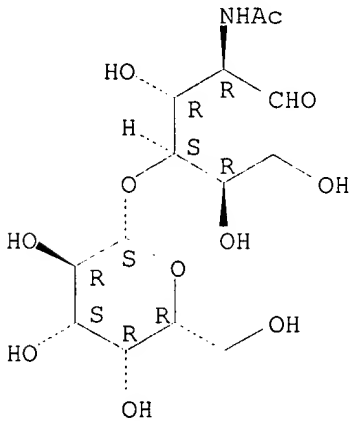
(1) Adelhorst, K; Carbohydr Res 1993, V242, P69 HCAPLUS
(2) Endo, T; Appl Microbiol Biotechnol 2000, V53, P257 HCAPLUS
(3) Endo, T; Carbohydr Res 1999, V316, P179 HCAPLUS
(4) Feizi, T; Nature 1985, V314, P53 HCAPLUS
(5) Fujio, T; Biosci Biotechnol Biochem 1997, V61, P956 HCAPLUS
(6) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(7) Hakomori, S; Adv Cancer Res 1989, V52, P257 HCAPLUS
(8) Hakomori, S; Histochem J 1992, V24, P771 HCAPLUS
(9) Ichikawa, Y; J Am Chem Soc 1992, V114, P9283 HCAPLUS
(10) Ilver, D; Science 1998, V279, P373 HCAPLUS
(11) Koizumi, S; Nat Biotechnol 1998, V16, P847 HCAPLUS
(12) Kretzschmar, G; Tetrahedron 1998, V54, P6341 HCAPLUS
(13) Lubineau, A; J Mol Catal B: Enzym 1998, V5, P229 HCAPLUS
(14) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(15) Mengin-Lecreulx, D; J Bacteriol 1996, V271, P32 HCAPLUS
(16) Nishi, T; Agric Biol Chem 1984, V48, P669 HCAPLUS
(17) Springer, T; Annu Rev Physiol 1995, V57, P827 HCAPLUS
(18) Stevenson, G; J Bacteriol 1996, V178, P4885 HCAPLUS
(19) Sturla, L; FEBS Lett 1997, V412, P126 HCAPLUS
(20) Tabata, K; Biotechnol Lett 2000, V22, P479 HCAPLUS
(21) Varki, A; Essentials of Glycobiology 1999
(22) Yamamoto, K; Agric Biol Chem 1984, V48, P823 HCAPLUS

IT **32181-59-2**, **N-Acetyl lactosamine**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(large-scale prodn. of GDP-**fucose** and Lewis X by bacterial coupling)

RN 32181-59-2 HCAPLUS

CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L117 ANSWER 4 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 2000:720765 HCAPLUS

DN 134:2450

TI Synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**

AU Rasko, David A.; Wang, Ge; Monteiro, Mario A.; Palcic, Monica M.; Taylor,

Diane E.
CS Department of Medical Microbiology and Immunology, Univ. of Alberta, Edmonton, AB, Can.
SO European Journal of Biochemistry (**2000**), 267(19), 6059-6066
CODEN: EJBCAI; ISSN: 0014-2956
PB Blackwell Science Ltd.
DT Journal
LA English
CC 10-2 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 7
AB The identification of **Helicobacter pylori** isolates that express exclusively type I Lewis antigens is necessary to det. the biosynthetic pathway of these antigens. Fast-atom bombardment MS provides evidence that the **H. pylori** isolate UA1111 expresses predominantly Leb, with H type I and Lea in lesser amts. Cloning and expression of the **H. pylori** **fucosyltransferases** (FucTs) allows comparisons with previously identified **H. pylori** enzymes and detn. of the enzyme specificities. Although all FucT, one .alpha.(1,2) FucT and two .alpha.(1,3/4) FucTs, appear to be functional in this isolate, their activities are lower and enzyme specificities are different to other **H. pylori** FucTs previously characterized. Studies of the cloned enzyme activities and mutational anal. indicate that Lea acts as the substrate for the synthesis of Leb. This is different from the human Leb biosynthetic pathway, but analogous to the biosynthetic pathway utilized by **H. pylori** for the prodn. of Ley.
ST Lewis blood group antigen formation **fucosyltransferase** Helicobacter; type I Lewis antigen formation Helicobacter
IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)
(**Lea**; synthesis of mono- and di-**fucosylated** type I **Lewis** blood group antigens by **Helicobacter pylori**)
IT **Blood-group substances**
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**Leb**; synthesis of mono- and di-**fucosylated** type I **Lewis** blood group antigens by **Helicobacter pylori**)
IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(futA; synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)
IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(futB; synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)
IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(futC; synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)
IT **Helicobacter pylori**
Mutation
(synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)
IT 37277-69-3, .alpha.(1,3/4) **Fucosyltransferase** **56093-23-3**, .alpha.-1,2 **Fucosyltransferase** 68247-53-0, .alpha.(1,3)-**Fucosyltransferase**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Amano, K; Clin Diag Lab Immunol 1997, V4, P540 HCAPLUS
(3) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(4) Appelmelk, B; Infect Immun 1999, V67, P5361 HCAPLUS
(5) Aspinall, G; Carbohyd Lett 1994, V1, P151 HCAPLUS
(6) Boren, T; Science 1993, V262, P1892 HCAPLUS
(7) Claeys, D; Gastroenterology 1998, V115, P340 HCAPLUS
(8) Edwards, N; Mol Microbiol 2000, V35, P1530 HCAPLUS
(9) Falk, P; Proc Natl Acad Sci 1993, V90, P2035 HCAPLUS
(10) Ge, Z; Annu Rev Microbiol 1999, V53, P353 HCAPLUS
(11) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(12) Henry, S; Vox Sang 1995, V69, P166 HCAPLUS
(13) Ilver, D; Science 1998, V279, P373 HCAPLUS
(14) Kukowska-Latallo, J; Gene Develop 1990, V4, P1288 HCAPLUS
(15) Logan, S; Mol Microbiol 2000, V35, P1156 HCAPLUS
(16) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(17) Monteiro, M; Eur J Biochem 2000, V276, P305
(18) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(19) Oriol, R; Glycobiology 1999, V9, P323 HCAPLUS
(20) Rasko, D; J Biol Chem 2000, V276, P4988
(21) Rasko, D; J Infect Dis 2000, V181, P1089 HCAPLUS
(22) Sambrook, J; Molecular cloning. A Laboratory Manual 1989
(23) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(24) Tabor, S; Proc Natl Acad Sci USA 1985, V82, P1074 HCAPLUS
(25) Taylor, D; Am J Clin Path 1987, V87, P49 MEDLINE
(26) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(27) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(28) Valkonen, K; Infect Immun 1997, V65, P916 HCAPLUS
(29) Wang, G; Microbiology 1999, V145, P3245 HCAPLUS
(30) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(31) Wang, Y; Gene 1990, V94, P23 HCAPLUS
(32) Weston, B; J Biol Chem 1992, V267, P4152 HCAPLUS
(33) Whitfield, C; Trend Microbiol 1995, V3, P178 MEDLINE
(34) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(35) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS
(36) Yokota, S; Infect Immun 2000, V68, P151 HCAPLUS

IT **56093-23-3**, .alpha.-1,2 **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(synthesis of mono- and di-**fucosylated** type I Lewis blood group antigens by **Helicobacter pylori**)

RN 56093-23-3 HCAPLUS

CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 5 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 2000:685450 HCAPLUS

DN 133:333843

TI Phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide

AU Appelmelk, Ben J.; Martino, M. Celeste; Veenhof, Eveline; Monteiro, Mario A.; Maaskant, Janneke J.; Negrini, Riccardo; Lindh, Frank; Perry, Malcolm; Del Giudice, Giuseppe; Vandenbroucke-Grauls, Christina M. J. E.

CS Department of Medical Microbiology, Vrije Universiteit, Medical School, Amsterdam, 1081 BT, Neth.

SO Infection and Immunity (**2000**), 68(10), 5928-5932
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 15-8 (Immunochemistry)

AB **Helicobacter pylori** NCTC 11637 lipopolysaccharide (LPS) expresses the human blood group antigens Lewis x (Lex), Ley, and H type I. In this report, we demonstrate that the H type I epitope displays high-frequency phase variation. One variant expressed Lex and Ley and no H type I as detd. by serol.; this switch was reversible. Insertional mutagenesis in NCTC 11637 of JHP563 (a poly(C) tract contg. an open reading frame homologous to glycosyltransferases) yielded a transformant with a serotype similar to the phase variant. Structural anal. of the NCTC 11637 LPS confirmed the loss of the H type I epitope. Sequencing of JHP563 in strains NCTC 11637, an H type I-neg. variant, and an H type I-pos. switchback variant showed a C14 (gene on), C13 (gene off), and C14 tract, resp. Inactivation of strain G27, which expresses Lex, Ley, H type I, and Lea, yielded a transformant that expressed Lex and Ley. We conclude that JHP563 encodes a .beta.3-galactosyltransferase involved in the biosynthesis of H type I and Lea and that phase variation in H type I is due to C-tract changes in this gene. A second H type I-neg. variant (variant 3a) expressed Lex and Lea and had lost both H type I and Ley expression. Inactivation of HP093-HP094 resulted in a transformant expressing Lex and lacking Ley and H type I. Structural anal. of a mutant LPS confirmed the serol. data. We conclude that the HP093-HP094 .alpha.2-**fucosyltransferase** (.alpha.2-FucT) gene product is involved in the biosynthesis of both Ley and Lex. Finally, we inactivated HP0379 in strain 3a. The transformant had lost both Lex and Lea expression, which demonstrates that the HP0379 gene product is both an .alpha.3- and an .alpha.4-FucT. Our data provide understanding at the mol. level of how **H. pylori** is able to diversify in the host, a requirement likely essential for successful colonization and transmission.

ST Helicobacter lipopolysaccharide blood group epitope

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(H; phase variation in H type I and **Lewis** a epitopes of **Helicobacter pylori** lipopolysaccharide)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Lea**; phase variation in H type I and **Lewis** a epitopes of **Helicobacter pylori** lipopolysaccharide)

IT Lipopolysaccharides
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(bacterial; phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide)

IT Epitopes
Helicobacter pylori
(phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide)

IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide formed by)

IT 39279-34-0 **56093-23-3** 111310-37-3
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide formed by)

RE.CNT 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Appelmelk, B; Gut 1999, V45(Suppl 3), PA23
(3) Appelmelk, B; Gut 2000, V47, P10 HCAPLUS
(4) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(5) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(6) Appelmelk, B; Infect Immun 1999, V67, P5361 HCAPLUS
(7) Appelmelk, B; Trends Microbiol 1997, V5, P70 MEDLINE
(8) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(9) Bijlsma, J; Infect Immun 1999, V67, P2433 HCAPLUS
(10) Blanchard, D; Rev Fr Transfus Hemobiol 1992, V35, P239 MEDLINE
(11) Censini, S; Proc Natl Acad Sci USA 1996, V93, P14648 HCAPLUS
(12) Edwards, N; Mol Microbiol 2000, V35, P1530 HCAPLUS
(13) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(14) Logan, S; Mol Microbiol 2000, V35, P1156 HCAPLUS
(15) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(16) Monteiro, M; Eur J Biochem 2000, V267, P305 HCAPLUS
(17) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(18) Rasko, D; J Biol Chem 2000, V275, P4988 HCAPLUS
(19) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(20) Van Dam, G; Eur J Biochem 1994, V225, P467 HCAPLUS
(21) Wang, G; Microbiology 1999, V145, P3245 HCAPLUS
(22) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(23) Weiser, J; Mol Microbiol 1998, V30, P767 HCAPLUS
IT **56093-23-3**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(phase variation in H type I and Lewis a epitopes of **Helicobacter pylori** lipopolysaccharide formed by)
RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 6 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:544056 HCAPLUS
DN 134:249290
TI Expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts: the propensity to express type 1 blood-group antigens
AU Monteiro, Mario A.; Zheng, Peng-Yuan; Ho, Bow; Yokota, Shin-Ichi; Amano, Ken-Ichi; Pan, Zhi-Jun; Berg, Douglas E.; Chan, Kenneth H.; MacLean, Leann L.; Perry, Malcolm B.
CS Institute for Biological Sciences, National Research Council, Ottawa, ON, K1A 0R6, Can.
SO Glycobiology (**2000**), 10(7), 701-713
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB Past studies have shown that the cell surface lipopolysaccharides (LPSs) of the ubiquitous human gastric pathogen **Helicobacter pylori** (a type 1 carcinogen) isolated from people residing in Europe and North America express predominantly type 2 Lewis x (Lex) and Ley epitopes and, infrequently, type 1 Lea, Leb, and Led antigens. This prodn. of Lewis blood-group structures by **H. pylori** LPSs, similar to those found in the surfaces of human gastric cells, allows the bacterium to mimic its human niche. In this study, LPSs of **H. pylori** strains extd. from patients living in China, Japan, and Singapore were chem. and serol. analyzed. When compared with

Western **H. pylori** LPSs, these Asian strains showed a stronger tendency to produce type 1 blood groups. Of particular interest, and novel observations in **H. pylori**, the O-chain regions of strains F-58C and R-58A carried type 1 Lea without the presence of type 2 Lex, strains R-7A and H607 were shown to have the capability of producing the type 1 blood group A antigen, and strains CA2, H507, and H428 expressed simultaneously the **difucosyl** isomeric antigens, type 1 Leb and type 2 Ley. The apparent proclivity for the prodn. of type 1 histo-blood group antigens in Asian **H. pylori** LPSs, as compared with Western strains, may be an adaptive evolutionary effect in that differences in the gastric cell surfaces of the resp. hosts might be significantly dissimilar to select for the formation of different LPS structures on the resident **H. pylori** strain.

ST Helicobacter lipopolysaccharide blood group antigen mimicry

IT Blood-group substances
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(A; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT Blood-group substances
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(B; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lea**; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Leb**; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT Blood-group substances
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(Led; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Ley**; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT Lipopolysaccharides
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(bacterial; expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

IT **Helicobacter pylori**
(expression of histo-blood group antigens by lipopolysaccharides of **Helicobacter pylori** strains from Asian hosts)

RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE
(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(3) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(4) Aspinall, G; Carbohydr Lett 1994, V1, P156
(5) Aspinall, G; Eur J Biochem 1997, V248, P592 HCAPLUS
(6) Aspinall, G; Glycobiology 1999, V9, P1235 HCAPLUS
(7) Ciucanu, I; Carbohydr Res 1984, V131, P209 HCAPLUS
(8) Davidson, J; Gastroenterology 1992, V103, P1552 MEDLINE
(9) Dell, A; Carbohydr Res 1990, V200, P59 HCAPLUS
(10) Dubois, M; Anal Chem 1956, V28, P350 HCAPLUS
(11) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(12) Egge, H; Mass Spectrom Rev 1987, V6, P331 HCAPLUS
(13) Gunner, S; Chem Ind 1961, P255 HCAPLUS
(14) Hakomori, S; Adv Cancer Res 1989, V52, P257 HCAPLUS
(15) Hanfland, P; Carbohydr Res 1988, V178, P1 HCAPLUS
(16) Kobayashi, K; Am J Gastroenterol 1993, V88, P919 MEDLINE
(17) Leotein, K; Carbohydr Res 1978, V62, P359
(18) Marshall, B; Lancet 1984, V8390, P1311
(19) Mollicone, R; Lab Invest 1985, V53, P219 MEDLINE
(20) Monteiro, M; Eur J Biochem 2000, V267, P305 HCAPLUS
(21) Monteiro, M; FEMS Microbiol Lett 1997, V154, P103 HCAPLUS
(22) Monteiro, M; Glycobiology 1998, V8, P107 HCAPLUS
(23) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(24) O'Croinin, T; Gastroenterology 1998, V114, P690
(25) Pohlentz, G; J Carbohydr Chem 1998, V17, P1151 HCAPLUS
(26) Sawardeker, J; Anal Chem 1965, V37, P1602 HCAPLUS
(27) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(28) Westphal, O; Methods Carbohydr Chem 1965, V5, P88
(29) Yokota, K; Infect Immun 1998, V66, P3006

L117 ANSWER 7 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:525066 HCAPLUS
DN 133:234795
TI Lewis antigens in **Helicobacter pylori**: biosynthesis and phase variation
AU Wang, Ge; Ge, Zhongming; Rasko, David A.; Taylor, Diane E.
CS Department of Medical Microbiology and Immunology, University of Alberta, Edmonton, AB, Can.
SO Molecular Microbiology (**2000**), 36(6), 1187-1196
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal; General Review
LA English
CC 10-0 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 15
AB A review with 47 refs. The lipopolysaccharides (LPS) of most **Helicobacter pylori** strains contain complex carbohydrates known as Lewis antigens that are structurally related to the human blood group antigens. Investigations on the genetic determinants involved in the biosynthesis of Lewis antigens have led to the identification of the **fucosyltransferases** of **H. pylori**, which have substrate specificities distinct from the mammalian **fucosyltransferases**. Compared with its human host, **H. pylori** utilizes a different pathway to synthesize the **difucosylated** Lewis antigens, Lewis y and Lewis b. Unique features in the **H. pylori fucosyltransferase** genes, including homopolymeric tracts mediating slipped-strand mispairing and the elements regulating translational frameshifting, enable **H. pylori** to produce variable LPS epitopes on its surface. These new findings have provided us with a basis to further examine the roles of mol. mimicry and phase variation of **H. pylori** Lewis antigen expression in both persistent infection and pathogenesis of this

important human gastric pathogen.
ST review Lewis antigen Helicobacter
IT **Blood-group substances**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation)
(**Le; Lewis** antigens in **Helicobacter pylori**: biosynthesis and phase variation)
IT **Helicobacter pylori**
(Lewis antigens in **Helicobacter pylori**: biosynthesis and phase variation)
RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(4) Appelmelk, B; Infect Immun 1999, V67, P5361 HCAPLUS
(5) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(6) Berg, D; Trends Microbiol 1997, V12, P468
(7) Boren, T; Science 1993, V262, P1892 HCAPLUS
(8) Chan, N; Glycobiology 1995, V5, P683 HCAPLUS
(9) Claeys, D; Gastroenterology 1998, V115, P340 HCAPLUS
(10) Faller, G; J Clin Pathol 1998, V51, P244 MEDLINE
(11) Ge, Z; Gastroenterology 1999, V116, PA169
(12) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(13) Gibson, J; Lett Appl Microbiol 1998, V26, P399 HCAPLUS
(14) Hakomori, S; Histochem J 1992, V24, P771 HCAPLUS
(15) Heneghan, M; FEMS Immunol Med Microbiol 1998, V20, P257 HCAPLUS
(16) Heneghan, M; Infect Immun 2000, V68, P937 HCAPLUS
(17) Henry, S; Vox Sang 1995, V69, P166 HCAPLUS
(18) Kleene, R; Biochim Biophys Acta 1993, V1154, P283 HCAPLUS
(19) Knirel, Y; Eur J Biochem 1999, V266, P123 HCAPLUS
(20) Kuipers, E; Aliment Pharmacol Ther 1997, V11(Suppl 1), P71
(21) Logan, S; Mol Microbiol 2000, V35, P1156 HCAPLUS
(22) Marshall, D; FEMS Immunol Med Microbiol 1999, V24, P79 HCAPLUS
(23) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(24) McGowan, C; Mol Microbiol 1998, V30, P19 HCAPLUS
(25) Monteiro, M; Eur J Biochem 2000, V267, P305 HCAPLUS
(26) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(27) Moran, A; FEMS Immnunol Med Microbiol 1995, V10, P271 HCAPLUS
(28) Moran, A; FEMS Immun Med Microbiol 1996, V16, P105 HCAPLUS
(29) Negrini, R; Gastroenterology 1996, V111, P655 MEDLINE
(30) Oriol, R; Glycobiology 1999, V9, P323 HCAPLUS
(31) Rasko, D; J Biol Chem 2000, V275, P4988 HCAPLUS
(32) Rasko, D; J Infect Dis 2000, V181, P1089 HCAPLUS
(33) Sherburne, R; Infect Immun 1995, V63, P4564 HCAPLUS
(34) Shiberu, B; Abstracts 10th International Workshop CHRO 1999, P130(HG8)
(35) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(36) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(37) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(38) Valkonen, K; Infect Immun 1997, V65, P916 HCAPLUS
(39) van Belkum, A; Microbiol Mol Biol Rev 1998, V62, P275 HCAPLUS
(40) Wang, G; Microbiology 1999, V145, P3245 HCAPLUS
(41) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(42) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(43) Wirth, H; Gastroenterology 1998, V114(Suppl 4), PA332
(44) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS
(45) Wirth, H; J Lab Clin Med 1999, V133, P488 MEDLINE
(46) Yokota, S; Infect Immun 1998, V66, P3006 HCAPLUS
(47) Yokota, S; Infect Immun 2000, V68, P151 HCAPLUS

L117 ANSWER 8 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:314847 HCAPLUS

DN 132:330636
TI Sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases
IN Taylor, Diane E.; Wang, Ge; Palcic, Monica
PA Governors of the University of Alberta, Can.
SO PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N015-54
ICS C12N009-10; C12P019-18; C07K016-40; C12Q001-48; G01N033-569; G01N033-574; C12Q001-68
CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 1, 7, 10, 15
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000026383	A1	20000511	WO 1999-CA1031	19991103 <--
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6238894	B1	20010529	US 1999-433598	19991102 <--
	EP 1127138	A1	20010829	EP 1999-953470	19991103 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002528122	T2	20020903	JP 2000-579755	19991103 <--
	US 2002037570	A1	20020328	US 2001-848838	20010503 <--
PRAI	US 1998-107268P	P	19981104 <--		
	US 1999-433598	A	19991102 <--		
	WO 1999-CA1031	W	19991103 <--		

OS MARPAT 132:330636
AB This invention provides protein and DNA sequences for a newly identified **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, which is involved in biosynthesis of **fucosylated** oligosaccharides including Lewis X, Lewis Y, Lewis B and H type 1, which are structurally similar to certain tumor-assocd. carbohydrate antigens found in mammals. The center region of fucT2 gene has a sequence of TAA repeats immediately following the poly C sequence, which are hypermutable and could offer an on-off mechanism for the expression of the gene, and changes of the repeat no. of the both tracts contribute to the variation of the fucT2 genotype in different strains. The invention further provides a method to measure the enzymic activity and acceptor specificity of .alpha.1,2-**fucosyltransferase**. The invention also relates to .alpha.1,2-**fucosyltransferase** antibodies which have research and diagnostic utility in the development of assays to detect mammalian tumors.
ST Helicobacter **fucosyltransferase** oligosaccharide lewis antigen biosynthesis cancer diagnosis
IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(H, for study enzyme activities of HpfucT2 in cytoplasmic and membrane fractions; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)
IT **Blood-group substances**

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(H, type 2; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Chimeric gene
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(HpfucT2 gene linked with a selectable marker gene; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Le**, B; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Le**, Y; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT **Blood-group substances**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Lex**, for study enzyme activities of HpfucT2 in cytoplasmic and membrane fractions; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Body fluid
(biol. fluid; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT DNA
RNA
cDNA
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(encoding HpfucT2; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Mutagenesis
(for study of .alpha.1,2-**fucosyltransferase** activities; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Oligosaccharides, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(**fucosylated**; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Neoplasm
(samples from malignant cells; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT DNA sequences
Diagnosis
Genetic vectors
Helicobacter pylori

Molecular cloning
Protein sequences
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Probes (nucleic acid)
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Antibodies
RL: ARG (Analytical reagent use); THU (Therapeutic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Fusion proteins (chimeric proteins)
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT Animal
Bacteria (Eubacteria)
Fungi
Plant (Embryophyta)
Yeast
(used as host cells for the expression of HpfucT2 protein; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT PCR (polymerase chain reaction)
(used for amplifying gene fucT2; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 616-91-1, NAC
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(LacNAC-R; as a substrate for HpfucT2 to produce **fucosylated** oligosaccharide; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 224432-11-5P
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(amino acid sequence; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 15839-70-0, GDP-**fucose**
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(for producing **fucosylated** oligosaccharide; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 221068-63-9
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU

(Occurrence); USES (Uses)
(nucleotide sequence; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 268534-70-9, 3: PN: WO0026383 PAGE: 30 unclaimed DNA 268534-71-0, 4: PN: WO0026383 PAGE: 30 unclaimed DNA 268534-72-1, 5: PN: WO0026383 PAGE: 30 unclaimed DNA 268534-73-2 268534-74-3 268535-77-9, 7: PN: WO0026383 SEQID: 6 unclaimed DNA
RL: PRP (Properties)
(unclaimed nucleotide sequence; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 268227-22-1 268227-23-2 268227-24-3 268227-25-4 268227-26-5
268227-27-6 268227-28-7 268227-29-8 268227-30-1 268227-31-2
268227-32-3 268227-33-4 268227-34-5 268227-35-6 268227-36-7
RL: PRP (Properties)
(unclaimed sequence; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

IT 9023-70-5, Glutamine synthetase
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(used as a selectable marker for the expression of HpfucT2 protein; sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Beyer, T; JOURNAL OF BIOLOGICAL CHEMISTRY 1980, V255(11), P5364 HCAPLUS
(2) Oomen, R; WO 9843478 HCAPLUS
(3) Oomen, R; WO 9843478 HCAPLUS
(4) Oomen, R; WO 9843478 A 1998 HCAPLUS
(5) Saunders, N; MOLECULAR MICROBIOLOGY 1998, V27(6), P1091 HCAPLUS
(6) Tomb, J; DATABASE EMBL 1997
(7) Wang, G; MICROBIOLOGY 1999, V145(11), P3245 HCAPLUS
(8) Wang, G; MOLECULAR MICROBIOLOGY 1999, V31(4), P1265 HCAPLUS

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); CAT (Catalyst use); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); USES (Uses)
(sequences of **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, and uses thereof in diagnosing disorders and in monitoring diseases)

RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 9 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:285204 HCAPLUS
DN 133:88095
TI Genotyping of cagA and vacA, Lewis antigen status, and analysis of the poly-(C) tract in the .alpha.(1,3)-**fucosyltransferase** gene of Irish **Helicobacter pylori** isolates

AU Ryan, K. A.; Moran, A. P.; Hynes, S. O.; Smith, T.; Hyde, D.; O'Morain, C. A.; Maher, M.
CS National Diagnostics Centre, BioResearch Ireland, Galway, Ire.
SO FEMS Immunology and Medical Microbiology (**2000**), 28(2), 113-120
CODEN: FIMIEV; ISSN: 0928-8244
PB Elsevier Science B.V.
DT Journal
LA English
CC 15-7 (Immunochemistry)
Section cross-reference(s): 3, 10
AB Much work has focused on trying to identify markers in **Helicobacter pylori** that might allow the eventual disease outcome of an infection to be predicted. In this study we examd. the cagA and vacA genotype, and Lewis status in a panel of 43 Irish **H. pylori** clin. isolates, and investigated a possible correlation with disease pathol. In addn., differences in the poly-(C) tract of the .alpha.(1,3)-**fucosyltransferase** gene were examd. to identify a possible correlation with gene expression. Only three of 43 isolates were cagA-neg., whereas the remaining 40 isolates, independent of pathol., were cagA-pos. In all the strains we examd., the vacA signal-sequence was type s1a. For the vacA mid-region 12/43 isolates were type m1 and 31/43 isolates were type m2. These data, and examn. of isolates from different pathol. groups, suggests that there is no correlation between virulence and vacA genotype in the Irish population of **H. pylori** isolates. Western blotting of whole cell lysates from 32 **H. pylori** isolates showed 3/32 displayed only the Lex epitope, 12/32 only the Ley, 13/32 both epitopes and 4/32 neither epitope. No apparent assocn. between Lewis phenotype and disease pathol. was evident. A range of lengths of poly-(C) tract were obsd. in the .alpha.(1,3)-**fucosyltransferase** gene, however the length of the tract in an isolate did not correlate with the Lewis structures present. We conclude that future studies on **H. pylori** pathogenesis should not alone focus on the importance of mol. markers, but also on the host response, including genetic background and immune responsiveness.
ST genotyping cagA vacA Lewis antigen **fucosyltransferase** gene Helicobacter
IT **Blood-group substances**
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(**Lex**; genotyping of cagA and vacA, **Lewis** antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)
IT **Blood-group substances**
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(**Ley**; genotyping of cagA and vacA, **Lewis** antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)
IT Gene, microbial
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(cagA; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)
IT Intestine, disease
(duodenum, ulcer; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-

fucosyltransferase gene of Irish humans' **Helicobacter pylori** isolates in)

IT Stomach, disease
(gastritis; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in)

IT Genotyping (method)
Helicobacter pylori
Virulence (microbial)
(genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)

IT Dyspepsia
(genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in)

IT Tumor markers
(genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in relation to)

IT Epitopes
(mapping; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)

IT Intestine, disease
(metaplasia; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in)

IT Esophagus
(reflux esophagitis; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in)

IT Stomach, disease
(ulcer; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates in)

IT Gene, microbial
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(vacA; genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)

IT 30811-80-4, Poly-(c) 68247-53-0, .alpha.(1,3)-**Fucosyltransferase**
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(genotyping of cagA and vacA, Lewis antigen status, and anal. of the poly-(C) tract in .alpha.(1,3)-**fucosyltransferase** gene of Irish humans' **Helicobacter pylori** isolates)

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Achtman, M; Mol Microbiol 1999, V32, P459 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Appelmelk, B; Infect Immun 1999, V67, P5361 HCAPLUS
(4) Atherton, J; Gastroenterology 1997, V112, P92 MEDLINE
(5) Atherton, J; J Biol Chem 1995, V270, P17771 HCAPLUS
(6) Berg, D; Trends Microbiol 1997, V5, P468 MEDLINE
(7) Campbell, S; Infect Immun 1997, V65, P3708 HCAPLUS

(8) Censini, S; Proc Natl Acad Sci USA 1996, V93, P14648 HCAPLUS
(9) Clayton, C; J Clin Microbiol 1992, V30, P192 HCAPLUS
(10) Covacci, A; Science 1999, V284, P1328 HCAPLUS
(11) Cover, T; J Biol Chem 1992, V267, P10570 HCAPLUS
(12) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(13) Evans, D; Helicobacter 1999, V4, P82 HCAPLUS
(14) Evans, D; J Clin Microbiol 1998, V36, P3435 HCAPLUS
(15) Faller, G; J Clin Pathol 1998, V51, P244 MEDLINE
(16) Fallone, C; Can J Gastroenterol 1999, V13, P251 MEDLINE
(17) Fujimoto, S; J Clin Microbiol 1994, V32, P331 HCAPLUS
(18) High, N; Mol Microbiol 1993, V9, P1275 HCAPLUS
(19) Hitchcock, P; J Bacteriol 1983, V154, P269 HCAPLUS
(20) Ito, Y; J Clin Microbiol 1997, V35, P1710 HCAPLUS
(21) Laemmli, U; Nature 1970, V227, P680 HCAPLUS
(22) Marshall, D; FEMS Immunol Med Microbiol 1995, V10, P317 HCAPLUS
(23) Marshall, D; FEMS Immunol Med Microbiol 1999, V24, P79 HCAPLUS
(24) Miehlke, S; Am J Gastroenterol 1996, V97, P1322
(25) Moran, A; FEMS Immunol Med Microbiol 1995, V10, P271 HCAPLUS
(26) Moran, A; Scand J Gastroenterol 1996, V31(Suppl 215), P22
(27) Pan, Z; J Clin Microbiol 1997, V35, P1344 MEDLINE
(28) Pan, Z; J Infect Dis 1998, V178, P220 HCAPLUS
(29) Rudi, J; J Clin Microbiol 1995, V36, P944
(30) Ryan, K; Gut 1999, V45(Suppl 111), P31
(31) Sherbourne, R; Infect Immun 1995, V63, P4564
(32) Strobel, S; J Clin Microbiol 1998, V36, P1285 HCAPLUS
(33) Takata, T; Am J Gastroenterol 1998, V93, P30 MEDLINE
(34) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(35) Towbin, H; Proc Natl Acad Sci USA 1979, V76, P4350 HCAPLUS
(36) van Doorn, L; Gastroenterology 1999, V116, P823 MEDLINE
(37) van Doorn, L; J Clin Microbiol 1998, V36, P2597 HCAPLUS
(38) van Doorn, L; J Clin Microbiol 1999, V37, P2306 HCAPLUS
(39) Vos, M; Electrophoresis 1999, V20, P1475
(40) Walsh, E; J Appl Microbiol 1997, V83, P67 HCAPLUS
(41) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(42) Wirth, H; Infect Immun 1996, V24, P4598
(43) Xiang, Z; Infect Immun 1995, V63, P94 HCAPLUS

L117 ANSWER 10 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:261855 HCAPLUS
DN 133:28309
TI Lewis X structures in the O antigen side-chain promote adhesion of **Helicobacter pylori** to the gastric epithelium
AU Edwards, Nicola J.; Monteiro, Mario A.; Faller, Gerhard; Walsh, Evelyn J.; Moran, Anthony P.; Roberts, Ian S.; High, Nicola J.
CS School of Biological Sciences, The University of Manchester, Manchester, M13 9PT, UK
SO Molecular Microbiology (**2000**), 35(6), 1530-1539
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB **Helicobacter pylori** NCTC11637 expresses a lipopolysaccharide (LPS) that comprises an O antigen side-chain with structural homol. to the human blood group antigen Lewis X (Lex). The role of this mol. in adhesion of **H. pylori** to gastric epithelial cells was investigated. Mutants expressing truncated LPS structures were generated through insertional mutagenesis of rfbM and galE; genes that encode GDP mannose pyrophosphorylase and galactose epimerase, resp. Compositional and structural anal. revealed that the galE mutant expressed a rough LPS that lacked an O antigen side-chain. In contrast, an O antigen side-chain was still synthesized by the rfbM mutant, but it lacked **fucose** and no longer reacted with anti-Lex

monoclonal antibodies (Mabs). The ability of these mutants to bind to paraffin-embedded sections from the antrum region of a human stomach was assessed. Adhesion of the wild type was characterized by tropic binding to the apical surface of mucosal epithelial cells and cells lining gastric pits. In contrast, both the rfbM and galE mutants failed to demonstrate tropic binding and adhered to the tissue surface in a haphazard manner. These results indicate that LPS and, more specifically, Lex structures in the O antigen side-chain play an important role in targeting **H. pylori** to specific cell lineages within the gastric mucosa. The role of Lex in this interaction was confirmed by the tropic binding of synthetic Lex, conjugated to latex beads, to gastric tissue. The obsd. pattern of adhesion was indistinguishable from that of wild-type **H. pylori**.

ST Lewis X O antigen adhesion Helicobacter stomach epithelium

IT Cell adhesion
Helicobacter pylori
Virulence (microbial)
(Lewis X structures in the O antigen side-chain promote adhesion of **Helicobacter pylori** to the gastric epithelium)

IT Lipopolysaccharides
O antigen
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(Lewis X structures in the O antigen side-chain promote adhesion of **Helicobacter pylori** to the gastric epithelium)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**Lex**; **Lewis** X structures in the O antigen side-chain promote adhesion of **Helicobacter pylori** to the gastric epithelium)

IT Stomach
(epithelium; Lewis X structures in the O antigen side-chain promote adhesion of **Helicobacter pylori** to the gastric epithelium)

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Adhya, S; Escherichia coli and Salmonella typhimurium: Cellular and Molecular Biology 1987, P1503 HCAPLUS
(2) Angstrom, J; Glycobiology 1998, V8, P297 HCAPLUS
(3) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(4) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(5) Appelmelk, B; Trends Microbiol 1997, V5, P70 MEDLINE
(6) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(7) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(8) Boren, T; Science 1993, V262, P1892 HCAPLUS
(9) Chan, N; Glycobiology 1995, V5, P683 HCAPLUS
(10) Ciucanu, I; Carbohydr Res 1984, V131, P209 HCAPLUS
(11) Dell, A; Carbohydr Res 1990, V200, P59 HCAPLUS
(12) Eggens, I; J Biol Chem 1989, V264, P9476 HCAPLUS
(13) Evans, D; Infect Immun 1993, V56, P2896
(14) Falk, P; Proc Natl Acad Sci USA 1993, V90, P2035 HCAPLUS
(15) Feizi, T; Nature 1985, V314, P53 HCAPLUS
(16) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(17) Guruge, J; Proc Natl Acad Sci USA 1998, V95, P3925 HCAPLUS
(18) Ilver, D; Science 1998, V279, P373 HCAPLUS
(19) Kannagi, R; J Biol Chem 1982, V257, P14865 HCAPLUS
(20) Kikuchi, S; Cancer 1995, V75, P2789 MEDLINE
(21) Kimura, A; Infect Immun 1986, V51, P60
(22) Larsen, E; Cell 1990, V63, P467 HCAPLUS
(23) Lesse, A; J Immunol Methods 1990, V126, P109 HCAPLUS
(24) Majewski, S; J Infect Dis 1988, V157, P465 HCAPLUS

(25) Marolda, C; J Bacteriol 1993, V175, P148 HCAPLUS
(26) McColl, K; J Infect 1997, V34, P7 MEDLINE
(27) McGee, D; Curr Top Microbiol Immunol 1999, V241, P155 HCAPLUS
(28) Moran, A; FEMS Immunol Med Microbiol 1995, V10, P271 HCAPLUS
(29) Moran, A; Scand J Gastroenterol 1996, V31(Suppl 215), P22
(30) Muotiala, A; Infect Immun 1992, V60, P1714 HCAPLUS
(31) Nathan, C; J Exp Biol 1981, V154, P1539 HCAPLUS
(32) Negrini, R; Gastroenterology 1991, V101, P437 MEDLINE
(33) Negrini, R; Gastroenterology 1996, V111, P655 MEDLINE
(34) Odenbreit, S; Mol Microbiol 1999, V31, P1537 HCAPLUS
(35) Sambrook, J; Molecular Cloning A Laboratory Manual 2nd edn 1989
(36) Sawardeker, J; Anal Chem 1965, V12, P1602
(37) Stoolman, L; Cell 1989, V56, P907 HCAPLUS
(38) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(39) Tomb, J; Science 1997, V388, P539 HCAPLUS
(40) Tsuda, M; Microbiol Immunol 1993, V37, P85 HCAPLUS
(41) Valkonen, K; Infect Immun 1994, V62, P3640 HCAPLUS
(42) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(43) Weiser, J; J Exp Med 1998, V187, P631 HCAPLUS
(44) Westphal, O; Methods Carbohydr Chem 1965, V5, P83 HCAPLUS
(45) Whitfield, C; Trends Microbiol 1995, V3, P178 MEDLINE
(46) Yang, Q; J Exp Med 1996, V183, P323 HCAPLUS

L117 ANSWER 11 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:148527 HCAPLUS
DN 132:290436
TI Cloning and characterization of the .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori**
AU Rasko, David A.; Wang, Ge; Palcic, Monica M.; Taylor, Diane E.
CS Department of Medical Microbiology and Immunology, University of Alberta, Edmonton, AB, T6G 2H7, Can.
SO Journal of Biological Chemistry (**2000**), 275(7), 4988-4994
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
CC 7-5 (Enzymes)
Section cross-reference(s): 3, 10, 15
AB The gastric pathogen **Helicobacter pylori** can express the histo blood group antigens, which are on the surface of many human cells. Most **H. pylori** strains express the type II carbohydrates, Lewis X and Y, whereas a small population express the type I carbohydrates, Lewis A and B. The expression of Lewis A and Lewis X, as in the case of **H. pylori** strain UA948, requires the addn. of **fucose** in .alpha.1,4 and .alpha.1,3 linkages to type I or type II carbohydrate backbones, resp. This work describes the cloning and characterization of a single **H. pylori** **fucosyltransferase** (FucT) enzyme, which has the ability to transfer **fucose** to both of the aforementioned linkages in a manner similar to the human **fucosyltransferase** V (Fuc-TV). Two homologous copies of the fucT gene have been identified in each of the genomes sequenced. The characteristic adenosine and cytosine tracts in the amino terminus and repeated regions in the carboxyl terminus are present in the DNA encoding the two UA948fucT genes, but these genes also contain differences when compared with previously identified **H. pylori** fucTs. The UA948fucTa gene encodes an approx. 52-kDa protein contg. 475 amino acids, whereas UA948fucTb does not encode a full-length FucT protein. In vitro, UA948FucTa appears to add **fucose** with a greater than 5-fold preference for type II chains but still retains significant activity using type I acceptors. The addn. of the **fucose** to the type II carbohydrate acceptors, by UA948FucTa, does not appear to be affected by **fucosylation** at other sites on the carbohydrate acceptor, but the rate of **fucose**

transfer is affected by terminal **fucosylation** of type I acceptors. Through mutational anal. we demonstrate that only FucTa is active in this **H. pylori** isolate and that inactivation of this enzyme eliminates expression of all Lewis antigens.

ST Helicobacter **fucosyltransferase** gene fucTa sequence; Lewis antigen **fucosyltransferase** gene fucTa Helicobacter

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Lea**; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of **Lewis** A and **Lewis** X antigens)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Lex**; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of **Lewis** A and **Lewis** X antigens)

IT DNA sequences
Helicobacter pylori
Protein sequences
(cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

IT Gene, microbial
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(fucTa; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

IT Gene, microbial
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(fucTb; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

IT 264253-21-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

IT 37277-69-3, .alpha.(1,3/4) **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

IT 256620-87-8, GenBank AF194963
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(nucleotide sequence; cloning and characterization of .alpha.(1,3/4) **fucosyltransferase** of **Helicobacter pylori** responsible for expression of Lewis A and Lewis X antigens)

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Amano, K; Clin Diag Lab Immunol 1997, V4, P540 HCAPLUS
(3) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(4) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(5) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(6) Breton, C; Glycobiology 1996, V6, Pvii HCAPLUS
(7) Breton, C; Glycobiology 1998, V8, P87 HCAPLUS

(8) Chan, N; Glycobiology 1995, V5, P683 HCAPLUS
(9) Claeys, D; Gastroenterology 1998, V115, P340 HCAPLUS
(10) Costa, J; J Biol Chem 1997, V272, P11613 HCAPLUS
(11) Costache, M; J Biol Chem 1997, V272, P29721 HCAPLUS
(12) Costache, M; Transfus Clin Biol 1997, V4, P367 MEDLINE
(13) de Vries, T; J Biol Chem 1995, V270, P8712 HCAPLUS
(14) Dupuy, F; J Biol Chem 1999, V274, P12257 HCAPLUS
(15) Elmgren, A; J Biol Chem 1997, V272, P21994 HCAPLUS
(16) Elmgren, A; Vox Sang 1996, V70, P97 HCAPLUS
(17) Gallet, P; Glycobiology 1998, V8, P919 HCAPLUS
(18) Ge, Z; Helicobacter pylori Protocols 1992, P145
(19) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(20) Hardy, E; Anal Biochem 1997, V244, P28 HCAPLUS
(21) Henry, S; Vox Sang 1995, V69, P166 HCAPLUS
(22) Kukowska-Latallo, J; Genes Dev 1990, V4, P1288 HCAPLUS
(23) Legault, D; J Biol Chem 1995, V270, P20987 HCAPLUS
(24) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(25) Mollicone, R; J Biol Chem 1994, V269, P20987 HCAPLUS
(26) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(27) Negrini, R; Gastroenterology 1996, V111, P655 MEDLINE
(28) Nishihara, S; Biochem Biophys Res Commun 1993, V196, P624 HCAPLUS
(29) Oriol, R; Glycobiology 1999, V9, P323 HCAPLUS
(30) Pang, H; Glycoconj J 1998, V15, P961 HCAPLUS
(31) Sambrook, J; Molecular Cloning: A Laboratory Manual, 2nd Ed 1989
(32) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(33) Tabor, S; Proc Natl Acad Sci U S A 1985, V82, P1074 HCAPLUS
(34) Taylor, D; Am J Clin Path 1987, V87, P49 MEDLINE
(35) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(36) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(37) Towbin, H; Proc Natl Acad Sci U S A 1979, V76, P4350 HCAPLUS
(38) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(39) Wang, Y; Gene (Amst) 1990, V34, P23
(40) Weston, B; J Biol Chem 1992, V267, P4152 HCAPLUS
(41) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS
(42) Xu, Z; J Biol Chem 1996, V271, P8818 HCAPLUS

L117 ANSWER 12 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:93957 HCAPLUS
DN 132:234079
TI Lipopolysaccharide structures of **Helicobacter pylori** genomic strains 26695 and J99, mouse model **H. pylori** Sydney strain, **H. pylori** P466 carrying **sialyl Lewis X**, and **H. pylori** UA915 expressing Lewis B. Classification of **H. pylori** lipopolysaccharides into glycotype families
AU Monteiro, Mario A.; Appelmelk, Ben J.; Rasko, David A.; Moran, Anthony P.; Hynes, Sean O.; MacLean, Leann L.; Chan, Ken H.; St Michael, Frank; Logan, Susan M.; O'Rourke, Jani; Lee, Adrian; Taylor, Diane E.; Perry, Malcolm B.
CS Institute for Biological Sciences, National Research Council, Ottawa, ON, Can.
SO European Journal of Biochemistry (**2000**), 267(2), 305-320
CODEN: EJBCAI; ISSN: 0014-2956
PB Blackwell Science Ltd.
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB This study describes the mol. makeup of the cell-wall lipopolysaccharides (LPSs) (O-chain polysaccharide.fwdarw.core oligosaccharide.fwdarw.lipid A) from 5 **H. pylori** strains: **H. pylori** 26695 and J99, the complete genome sequences of which have been published, the established mouse model Sydney strain (SS1), and the symptomatic strains P466 and UA915. All chem. and serol. expts. were performed on the intact LPSs. **H. pylori** 26695 and SS1 possessed either

a low-Mr semi-rough-form LPS carrying mostly a single Ley type-2 blood-group determinant in the O-chain region covalently attached to the core oligosaccharide or a high-Mr smooth-form LPS, as did strain J99, with an elongated partially **fucosylated** type-2 **N-acetyllactosamine** (polyLacNAc) O-chain polymer, terminated mainly by a Lex blood-group determinant, connected to the core oligosaccharide. In the midst of semi-rough-form LPS glycoforms, **H. pylori** 26695 and SS1 also expressed in the O-chain region a **difucosylated** antigen, .alpha.-L-Fucp(1-3)-.alpha.-L-Fucp(1-4)-.beta.-D-GlcpNAc, and the cancer-cell-related type-1 or type-2 linear B-blood-group antigen, .alpha.-D-Galp(1-3)-.beta.-D-Galp(1-3 or 4)-.beta.-D-GlcpNAc. The LPS of **H. pylori** strain P466 carried the cancer-assocd. type-2 **sialyl Lex** blood-group antigen, and the LPS from strain UA915 expressed a type-1 Leb blood-group unit. These findings should aid investigations that focus on identifying and characterizing genes responsible for LPS biosynthesis in genomic strains 26695 and J99, and in understanding the role of **H. pylori** LPS in animal model studies. The LPSs from the **H. pylori** strains studied to date were grouped into specific glycotype families.

ST lipopolysaccharide Helicobacter

IT **Helicobacter pylori**
(lipopolysaccharide structures of **Helicobacter pylori**
)

IT Lipopolysaccharides
Oligosaccharides, properties
RL: PRP (Properties)
(lipopolysaccharide structures of **Helicobacter pylori**
)

RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Akopyants, N; Helicobacter 1997, V2, P48 MEDLINE
(2) Alkout, A; Gastroenterology 1997, V112, P1179 HCAPLUS
(3) Alm, R; Nature (London) 1999, V397, P176 HCAPLUS
(4) Amano, K; Microbiol Immunol 1998, V42, P509 HCAPLUS
(5) Appelmelk, B; Infec Immun 1999, V67, P5361 HCAPLUS
(6) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(7) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(8) Aspinall, G; Carbohydr Lett 1994, V1, P156
(9) Aspinall, G; Eur J Biochem 1997, V248, P592 HCAPLUS
(10) Aspinall, G; Ir J Med Sci 1997, V166(Suppl 3), P26
(11) Bliss, C; Infect Immun 1998, V66, P5357 HCAPLUS
(12) Boren, T; Science 1993, V262, P1892 HCAPLUS
(13) Cammarota, G; Ital J Gastroenterol Hepatol 1998, V30(Suppl 3), PS304
(14) Danesh, J; Br Med J 1998, V316, P1130 MEDLINE
(15) Dell, A; Carbohydr Res 1990, V200, P59 HCAPLUS
(16) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(17) Egge, H; Mass Spectrom Rev 1987, V6, P331 HCAPLUS
(18) Falk, P; Microbiol Mol Biol Rev 1998, V62, P1157 MEDLINE
(19) Gibson, J; Lett Appl Microbio 1998, V26, P399 HCAPLUS
(20) Hakomori, S; Adv Cancer Res 1989, V52, P257 HCAPLUS
(21) Hanfland, P; Carbohydr Res 1988, V178, P1 HCAPLUS
(22) Kobayashi, K; Am J Gastroenterol 1993, V88, P919 MEDLINE
(23) Lee, A; Br Med Bull 1998, V54, P163 MEDLINE
(24) Lee, A; Gastroenterology 1997, V112, P1386 MEDLINE
(25) McGowan, C; Mol Microbiol 1998, V30, P19 HCAPLUS
(26) Mendall, M; Br Heart J 1994, V71, P437 MEDLINE
(27) Mollicone, R; Lab Invest 1985, V53, P219 MEDLINE
(28) Monteiro, M; FEMS Microbiol Lett 1997, V154, P103 HCAPLUS
(29) Monteiro, M; Glycobiology 1998, V8, P107 HCAPLUS
(30) Monteiro, M; Ir J Med Sci 1997, V166(Suppl 3), P61
(31) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(32) Moran, A; J Bacteriol 1997, V179, P6453 HCAPLUS

(33) Murray, L; Cardiologia 1997, V42, P1027 MEDLINE
(34) Osaki, T; J Med Microbiol 1998, V47, P505 HCAPLUS
(35) O'Croinin, T; Gastroenterology 1998, V114, P690
(36) Piotrowski, J; J Physiol Pharmacol 1998, V49, P3 HCAPLUS
(37) Pohlentz, G; J Carbohydr Chem 1998, V17, P1151 HCAPLUS
(38) Rietschel, E; Curr Top Microbiol Immunol 1996, V216, P39 HCAPLUS
(39) Sheu, B; Dig Dis Sci 1999, V44, P868 MEDLINE
(40) Slomiany, B; Scand J Gastroenterol 1998, V33, P916 HCAPLUS
(41) Strecker, G; Eur J Biochem 1992, V207, P995 HCAPLUS
(42) Suda, Y; J Biochem (Tokyo) 1997, V121, P1129 HCAPLUS
(43) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(44) Tomb, J; Nature (London) 1997, V388, P539 HCAPLUS
(45) van Doorn, D; Clin Exp Immunol 1999, V115, P421
(46) Walsh, E; Ir J Med Sci 1997, V166(Suppl 3), P27
(47) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(48) Whitfield, C; Trends Microbiol 1995, V3, P178 MEDLINE

L117 ANSWER 13 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:745409 HCAPLUS
DN 132:62834
TI Novel **Helicobacter pylori** .alpha.1,2-**fucosyltransferase**, a key enzyme in the synthesis of Lewis antigens
AU Wang, Ge; Boulton, Peter G.; Chan, Nora W. C.; Palcic, Monica M.; Taylor, Diane E.
CS Departments of Medical Microbiology and Immunology, University of Alberta, Edmonton, AB, T6G 2H7, Can.
SO Microbiology (Reading, United Kingdom) (**1999**), 145(11), 3245-3253
CODEN: MROBEO; ISSN: 1350-0872
PB Society for General Microbiology
DT Journal
LA English
CC 15-2 (Immunochemistry)
AB **Helicobacter pylori** lipopolysaccharides (LPS) contain complex carbohydrates known as Lewis antigens which may contribute to the pathogenesis and adaptation of the bacterium. Involved in the biosynthesis of Lewis antigens is an .alpha.1,2-**fucosyltransferase** (FucT) that adds **fucose** to the terminal .beta.Gal unit of the O-chain of LPS. Recently, the **H. pylori** (Hp) .alpha.1,2-FucT-encoding gene (fucT2) was cloned and analyzed in detail. However, due to the low level of expression and instability of the protein, its enzymic activity was not demonstrated. In this study, the Hp fucT2 gene was successfully overexpressed in Escherichia coli. Sufficient amts. of the protein were obtained which revealed .alpha.1,2-**fucosyltransferase** activity to be assocd. with the protein. A series of substrates were chosen to examine the acceptor specificity of Hp .alpha.1,2-FucT, and the enzyme reaction products were identified by capillary electrophoresis. In contrast to the normal mammalian .alpha.1,2-FucT (H or Se enzyme), Hp .alpha.1,2-FucT prefers to use Lewis X [.beta.Gal1-4(.alpha.Fuc1-3).beta.GlcNAc] rather than LacNAc [.beta.Gal1-4.beta.GlcNAc] as a substrate, suggesting that **H. pylori** uses a novel pathway (via Lewis X) to synthesize Lewis Y. Hp .alpha.1,2-FucT also acts on type 1 acceptor [.beta.Gal1-3.beta.GlcNAc] and Lewis a [.beta.Gal1-3(.alpha.Fuc1-4).beta.GlcNAc], which provides **H. pylori** with the potential to synthesize H type 1 and Lewis b epitopes. The ability to transfer **fucose** to a **monofucosylated** substrate (Lewis X or Lewis a) makes Hp .alpha.1,2-FucT distinct from normal mammalian .alpha.1,2-FucT.
ST Helicobacter **fucosyltransferase** Lewis antigen **fucose**
IT **Helicobacter pylori**
(**H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); PROC (Process)
(**Le; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of **Lewis** antigens)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**Lea; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of **Lewis** antigens)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); PROC (Process)
(**Leb; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of **Lewis** antigens)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(**Lex; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of **Lewis** antigens)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BPN (Biosynthetic preparation); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PREP (Preparation); PROC (Process)
(**Ley; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of **Lewis** antigens)

IT Gene, microbial
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(for .alpha.1,2-**fucosyltransferase; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

IT Galactosylation
(**fucosylation; H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(**H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

IT 3615-37-0, D-**Fucose**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Alm, R; Nature 1999, V397, P176 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(4) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(5) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(6) Avent, N; Brit J Biomed Sci 1997, V54, P16 HCAPLUS
(7) Blaszczyk-Thurin, M; Biochem Biophys Res Commun 1988, V151, P100 HCAPLUS
(8) Chan, N; Glycobiology 1995, V5, P683 HCAPLUS
(9) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(10) Hakomori, S; Adv Cancer Res 1989, V52, P257 HCAPLUS

(11) Herry, S; Vox Sang 1995, V69, P166
(12) Hitoshi, S; J Biol Chem 1996, V271, P16975 HCAPLUS
(13) Kannagi, R; Glycoconj J 1997, V14, P577 HCAPLUS
(14) Kleene, R; Biochim Biophys Acta 1993, V1154, P283 HCAPLUS
(15) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(16) Miyake, M; Biochemistry 1991, V30, P3328 HCAPLUS
(17) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(18) Palcic, M; Glycoconj J 1988, V5, P49 HCAPLUS
(19) Sakamoto, J; Cancer Res 1986, V46, P1553 HCAPLUS
(20) Sambrook, J; Molecular Cloning:a Laboratory Manual, 2nd edn 1989
(21) Sarnesto, A; J Biol Chem 1990, V265, P15067 HCAPLUS
(22) Sarnesto, A; J Biol Chem 1992, V267, P2737 HCAPLUS
(23) Sherburne, R; Infect Immun 1995, V63, P4564 HCAPLUS
(24) Sun, J; Proc Natl Acad Sci USA 1995, V92, P5724 HCAPLUS
(25) Tabor, S; Proc Natl Acad Sci USA 1985, V82, P1074 HCAPLUS
(26) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(27) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(28) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(29) Watkins, W; Glycoproteins 1995, P313 HCAPLUS
(30) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(31) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS
(32) Wirth, H; J Lab Clin Med 1999, V133, P488 MEDLINE
(33) Yao, Z; J Bacteriol 1992, V174, P7500 HCAPLUS
(34) Yazawa, S; Jpn J Cancer Res 1993, V84, P989 HCAPLUS

IT **56093-23-3P**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PREP (Preparation)
(**H. pylori** .alpha.1,2-**fucosyltransferase** and synthesis of Lewis antigens)

RN 56093-23-3 HCAPLUS

CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 14 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 1999:742453 HCAPLUS

DN 132:90454

TI Structural studies on lipopolysaccharides of serologically non-typable strains of **Helicobacter pylori**, AF1 and 007, expressing Lewis antigenic determinants

AU Knirel, Yuriy A.; Kocharova, Nina A.; Hynes, Sean O.; Widmalm, Goran; Andersen, Leif P.; Jansson, Per-Erik; Moran, Anthony P.

CS Karolinska Institute, Clinical Research Center, Huddinge University Hospital, Huddinge, S-141 86, Swed.

SO European Journal of Biochemistry (**1999**), 266(1), 123-131
CODEN: EJBCAI; ISSN: 0014-2956

PB Blackwell Science Ltd.

DT Journal

LA English

CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 15

AB In contrast to other **Helicobacter pylori** strains, which have serol. detectable Lewisx (Lex) and Lewisy (Ley) antigenic determinants in the O-specific polysaccharide chains of the lipopolysaccharides, **H. pylori** AF1 and 007 were non-typable with anti-Lex and anti-Ley antibodies. The carbohydrate portions of the lipopolysaccharides were liberated by mild acid hydrolysis and subsequently studied by sugar and methylation analyses, 1H-NMR spectroscopy, and electrospray ionization-mass spectrometry. Compared with each other, and with lipopolysaccharides of strains studied previously, the lipopolysaccharides of both AF1 and 007 showed

similarities, but also differences, in the structures of the core region and O-specific polysaccharide chains. The O-specific polysaccharide chains of both strains consisted of a short or long **polyfucosylated** poly-N-acetyl-.beta.-lactosamine chains, which were distinguished from those of other strains by a high degree of **fucosylation** producing a polymeric Lex chain terminating with Lex or Ley units. Where n = 0 or 1 in strain AF1 and 0 in strain 007, m = 0-2, 6-7 in strain AF1 and m = 0-2, 6-7 or .apprxeq. 40 in strain 007, the medium-size species being predominant. Therefore, compared with other strains, the lack of reactivity of lipopolysaccharide of **H. pylori** AF1 and 007 with anti-Lex and anti-Ley may reflect the presence of a polymeric Lex chain and has important implications for serol. and pathogenesis studies. As the substitution pattern of a D-glycero-D-manno-heptose residue in the outer core varied in the two strains, and an extended DD-heptan chain was present in some lipopolysaccharide species but not in others, this region was less conservative than the inner core region. The inner core L-glycero-D-manno-heptose region of both strains carried a 2-aminoethyl phosphate group, rather than a phosphate group, as reported previously for other **H. pylori** strains.

ST lipopolysaccharide structure Helicobacter nontypable Lewis antigenic determinant

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; structural studies on lipopolysaccharides of serol. nontypable **Helicobacter pylori** AF1 and 007 expressing **Lewis** antigenic determinants)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Ley**; structural studies on lipopolysaccharides of serol. nontypable **Helicobacter pylori** AF1 and 007 expressing **Lewis** antigenic determinants)

IT Epitopes
Helicobacter pylori
(structural studies on lipopolysaccharides of serol. nontypable **Helicobacter pylori** AF1 and 007 expressing Lewis antigenic determinants)

IT O antigen
Oligosaccharides, biological studies
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(structural studies on lipopolysaccharides of serol. nontypable **Helicobacter pylori** AF1 and 007 expressing Lewis antigenic determinants)

IT Lipopolysaccharides
RL: PRP (Properties)
(structural studies on lipopolysaccharides of serol. nontypable **Helicobacter pylori** AF1 and 007 expressing Lewis antigenic determinants)

RE.CNT 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Appelmelk, B; Immunol Today 1998, V19, P296 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Appelmelk, B; Trends Microbiol 1997, V5, P70 MEDLINE
(4) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(5) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(6) Aspinall, G; Eur J Biochem 1997, V248, P592 HCAPLUS
(7) Aspinall, G; Ir J Med Sci 1997, V166(Suppl 3), P26
(8) Aspinall, G; Pathogenesis and Host Response in Helicobacter pylori Infections 1997, P34
(9) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS

(10) Hakomori, S; J Biochem 1964, V55, P205 MEDLINE
(11) Hunt, R; Scand J Gastroenterol 1996, V31(Suppl 220), P3
(12) Jansson, P; Chem Commun University of Stockholm 1976, V8, P1
(13) Laemmli, U; Nature 1970, V227, P680 HCAPLUS
(14) Leontein, K; Carbohydr Res 1978, V62, P359 HCAPLUS
(15) Marshall, D; FEMS Immunol Med Microbiol 1999, V24, P79 HCAPLUS
(16) Monteiro, M; Glycobiology 1998, V8, P107 HCAPLUS
(17) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(18) Moran, A; FEMS Immunol Med Microbiol 1995, V10, P271 HCAPLUS
(19) Moran, A; J Bacteriol 1992, V174, P1370 HCAPLUS
(20) Moran, A; J Endotoxin Res 1996, V3, P521 HCAPLUS
(21) Moran, A; Scand J Gastroenterol 1996, V31(Suppl 215), P22
(22) Sawardeker, J; Anal Chem 1965, V37, P1602 HCAPLUS
(23) Schwarzmann, G; Carbohydr Res 1974, V34, P161 HCAPLUS
(24) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(25) Towbin, H; Proc Natl Acad Sci USA 1979, V76, P4350 HCAPLUS
(26) Tsai, C; Anal Biochem 1982, V119, P115 HCAPLUS
(27) Westphal, O; Methods Carbohydr Chem 1965, V5, P83 HCAPLUS
(28) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS

L117 ANSWER 15 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:600981 HCAPLUS
DN 131:284707
TI Altered mRNA expression of glycosyltransferases in human gastric carcinomas
AU Petretti, T.; Schulze, B.; Schlag, P. M.; Kemmner, W.
CS Department of Surgery and Surgical Oncology, Klinikum Charite, Robert-Rossle-Klinik at the Max-Delbruck-Center of Molecular Medicine, Berlin, D-13125, Germany
SO Biochimica et Biophysica Acta (**1999**), 1428(2-3), 209-218
CODEN: BBACAQ; ISSN: 0006-3002
PB Elsevier Science B.V.
DT Journal
LA English
CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3
AB Biosynthesis of carbohydrate structures is tissue-specific and developmentally regulated by glycosyltransferases like **fucosyl**-, sialyl- and N-acetylglucosaminyltransferases. During carcinogenesis, aberrant glycosylation leads to the development of tumor subpopulations with different adhesion properties. The aim of this contribution was to directly compare mRNA expression of several glycosyltransferases in surgical specimens of gastric carcinomas. Carcinoma specimens were classified and characterized according to the WHO/UICC system. In each case, the expression of 12 glycosyltransferase enzymes was studied simultaneously by RT-PCR. For semi-quant. anal., amplification of the sample sequence was compared with that of .beta.-actin, co-amplified within the same tube. Expression of N-acetylglucosaminyltransferase V in gastric carcinomas was significantly enhanced compared to normal tissue. Also, expression of sialyltransferase ST3Gal-IV and **fucosyltransferase** FT-IV was significantly enhanced in carcinoma tissue. No significant differences in glycosyltransferase expression were found in samples pos. for **Helicobacter pylori** or between the different gastric regions. Thus, carcinogenesis is characterized by specific alterations in mRNA expression of several glycosyltransferases. Future studies will show whether RT-PCR detection of the expression of these enzymes could be helpful for prognostic purposes.
ST glycosyltransferase mRNA stomach carcinoma
IT Stomach, neoplasm
(adenocarcinoma; altered mRNA expression of glycosyltransferases in human gastric carcinomas)
IT mRNA

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT Stomach, neoplasm
(carcinoma, metastasis; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT Stomach, neoplasm
(carcinoma; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT Gene
(expression; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT Stomach, neoplasm
(signet-ring cell carcinoma; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT 9075-81-4
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT 37277-69-3, **Fucosyltransferase** III 39279-34-0 60202-12-2, Sialyltransferase IV 68247-53-0, **Fucosyltransferase** IV 83588-90-3, N-Acetylglucosaminyltransferase V **125752-90-1**, Sialyltransferase III
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT **56093-23-3**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human H blood group; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

IT 9031-68-9, Galactosyltransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(isoforms; altered mRNA expression of glycosyltransferases in human gastric carcinomas)

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Bosch, J; Cancer Detect Prev 1998, V22, P319 MEDLINE
(2) Chomczynski, P; Anal Biochem 1987, V162, P156 HCAPLUS
(3) Dall'Olio, F; Int J Cancer 1989, V44, P434 MEDLINE
(4) Dall'Olio, F; Int J Cancer 1992, V50, P325 HCAPLUS
(5) Demetriou, M; J Cell Biol 1995, V130, P383 HCAPLUS
(6) Dennis, J; Semin Cancer Biol 1991, V2, P411 HCAPLUS
(7) Dohi, T; Cancer 1994, V73, P1552 HCAPLUS
(8) Fernandes, B; Cancer Res 1991, V51, P718 MEDLINE
(9) Gessner, P; Cancer Lett 1993, V75, P143 HCAPLUS
(10) Grundmann, U; Nucleic Acids Res 1994, V18, P667
(11) Hiraiwa, N; J Biol Chem 1996, V271, P31556 HCAPLUS
(12) Ikeda, Y; J Surg Oncol 1996, V62, P171 MEDLINE
(13) Ito, H; Int J Cancer 1997, V71, P556 HCAPLUS
(14) Kemmner, W; Clin Exp Metastasis 1994, V12, P245 HCAPLUS
(15) Kitagawa, H; Biochem Biophys Res Commun 1993, V194, P375 HCAPLUS
(16) Kitagawa, H; J Biol Chem 1994, V269, P1394 HCAPLUS
(17) Korczak, B; Adv Exp Med Biol 1994, V353, P95 MEDLINE
(18) Kudo, T; Lab Invest 1998, V78, P797 HCAPLUS
(19) Li, W; Am J Pathol 1994, V145, P470 HCAPLUS
(20) Masri, K; Biochem Biophys Res Commun 1998, V157, P663
(21) Morgenthaler, J; Biochem Biophys Res Commun 1990, V171, P860 HCAPLUS
(22) Natsuka, S; Curr Opin Struct Biol 1994, V4, P683 HCAPLUS
(23) Natsuka, S; J Biol Chem 1994, V269, P16789 HCAPLUS
(24) O'Hanlon, T; J Biol Chem 1989, V264, P17389 HCAPLUS
(25) Paulson, J; J Biol Chem 1989, V264, P10931 HCAPLUS

(26) Pilatte, Y; Glycobiology 1993, V3, P201 HCAPLUS
(27) Ponte, P; Nucleic Acids Res 1998, V12, P1687
(28) Saito, H; Biochem Biophys Res Commun 1994, V198, P318 HCAPLUS
(29) Sasaki, K; J Biol Chem 1993, V268, P22782 HCAPLUS
(30) Seelentag, W; Cancer Res 1998, V58, P5559 HCAPLUS
(31) Sun, J; Proc Natl Acad Sci USA 1995, V92, P5724 HCAPLUS
(32) Taniguchi, N; Glycobiology 1996, V6, P691 HCAPLUS
(33) Whitehouse, C; J Cell Biol 1997, V137, P1229 HCAPLUS
(34) Yago, K; Cancer Res 1993, V53, P5559 HCAPLUS

IT **125752-90-1**, Sialyltransferase III
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(altered mRNA expression of glycosyltransferases in human gastric carcinomas)
RN 125752-90-1 HCAPLUS
CN Sialyltransferase, cytidine monophosphoacetylneuraminate-lactosylceramide .alpha.2,3- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **56093-23-3**, .alpha.1.fwdarw.2 **Fucosyltransferase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(human H blood group; altered mRNA expression of glycosyltransferases in human gastric carcinomas)
RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 16 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:159079 HCAPLUS
DN 130:333527
TI Molecular genetic basis for the variable expression of Lewis Y antigen in **Helicobacter pylori**: analysis of the .alpha.(1,2) **fucosyltransferase** gene
AU Wang, Ge; Rasko, David A.; Sherburne, Richard; Taylor, Diane E.
CS Department of Medical Microbiology and Immunology, University of Alberta, Edmonton, AB, T6G 2H7, Can.
SO Molecular Microbiology (**1999**), 31(4), 1265-1274
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
CC 3-3 (Biochemical Genetics)
Section cross-reference(s): 7, 10, 15
AB **Helicobacter pylori** lipopolysaccharides (LPS) express human oncofetal antigens Lewis X and Lewis Y. The synthesis of Lewis Y involves the actions of .alpha.(1,3) and .alpha.(1,2) **fucosyltransferases** (FucTs). Here, we report the mol. cloning and characterization of genes encoding **H. pylori** .alpha.(1,2) FucT (Hp fucT2) from various **H. pylori** strains. We constructed Hp fucT2 knock-out mutants and demonstrated the loss of Lewis Y prodn. in these mutants by ELISA and immunoelectron microscopy. The Hp fucT2 gene contains a hypermutable sequence [poly(C) and TAA repeats], which provides a possibility of frequent shifting into and out of coding frame by a polymerase slippage mechanism. Thus, the Hp fucT2 gene displays two major genotypes, consisting of either a single full-length open reading frame (ORF; as in the strain UA802) or truncated ORFs (as in the strain 26695). In vitro expression of Hp fucT2 genes demonstrated that both types of the gene have the potential to produce the full-length protein. The prodn. of the full-length protein by the 26695 fucT2 gene could be attributed to translational -1 frameshifting, as a perfect translation frameshift cassette resembling that of the Escherichia coli dnaX gene is present. Examn. of the strain UA1174 revealed that its

fucT2 gene has a frameshifted ORF at the DNA level, which cannot be compensated by translation frameshifting, accounting for its Lewis Y off phenotype. In another strain, UA1218, the fucT2 gene is apparently turned off because of the loss of its promoter. Based on these data, we proposed a model for the variable expression of Lewis Y by **H. pylori**, in which regulation at the level of replication slippage (mutation), transcription and translation of the fucT2 gene may all be involved.

ST Lewis Y antigen Helicobacter **fucosyltransferase** gene; sequence gene fucT2 **fucosyltransferase** Helicobacter

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**Ley**; variable expression of **Ley** antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT Gene
(expression; variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT Gene, microbial
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(fucT2; variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT DNA sequences
Helicobacter pylori
Mutagenesis
Mutation
Protein sequences
Ribosomal frameshifting
Transcription, genetic
(variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT Promoter (genetic element)
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT 224432-11-5 224432-12-6 224432-13-7 224432-14-8 224432-16-0 224432-18-2 224432-19-3 224432-20-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(amino acid sequence; variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT 221068-63-9, GenBank AF076779 223658-12-6, GenBank AF093828 223658-13-7, GenBank AF093829 223658-14-8, GenBank AF093830 223658-15-9, GenBank AF093831 223658-16-0, GenBank AF093832 223658-17-1, GenBank AF093833
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(nucleotide sequence; variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)

IT **56093-23-3**, .alpha.-(1.fwdarw.2)-L-**Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-

fucosyltransferase gene)
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(2) Appelmelk, B; Infect Immun 1998, V66, P70 HCAPLUS
(3) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(4) Avent, N; Br J Biomed Sci 1997, V54, P16 HCAPLUS
(5) Berg, D; Trends Microbiol 1997, V12, P468
(6) Breton, C; Glycobiology 1998, V8, P87 HCAPLUS
(7) Cover, T; Adv Int Med 1996, V41, P85 MEDLINE
(8) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(9) Farabaugh, P; Annu Rev Genet 1996, V30, P507 HCAPLUS
(10) Flower, A; Proc Natl Acad Sci USA 1990, V87, P3713 HCAPLUS
(11) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(12) Ge, Z; Methods in Molecular Medicine 1997, P145 HCAPLUS
(13) Hakomori, S; Adv Cancer Res 1989, V52, P257 HCAPLUS
(14) Hitchcock, P; J Bacteriol 1983, V154, P269 HCAPLUS
(15) Kelly, R; J Biol Chem 1995, V270, P4640 HCAPLUS
(16) Kleene, R; Biochim Biophys Acta 1993, V1154, P283 HCAPLUS
(17) Kuipers, E; Alimen Pharmacol Therapeut 1997, V11(Suppl 1), P71
(18) Larsen, B; J Bacteriol 1994, V176, P6842 MEDLINE
(19) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(20) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(21) Roche, R; Trends Microbiol 1995, V3, P304 MEDLINE
(22) Saunders, N; Mol Microbiol 1998, V27, P1091 HCAPLUS
(23) Sherburne, R; Infect Immun 1995, V63, P4564 HCAPLUS
(24) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(25) Tsuchihashi, Z; Genes Dev 1992, V6, P511 HCAPLUS
(26) Van Kranenburg, R; Mol Microbiol 1997, V24, P387 HCAPLUS
(27) Van Putten, J; Mol Microbiol 1995, V16, P847 HCAPLUS
(28) Wang, Y; Gene 1990, V94, P23 HCAPLUS
(29) Watkins, W; Glycoproteins 1995, P313 HCAPLUS
(30) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(31) Wirth, H; Gastroenterology 1997, V112, PA331
(32) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS
(33) Zhang, L; Mol Microbiol 1997, V23, P63 HCAPLUS
IT **56093-23-3**, .alpha.-(1.fwdarw.2)-L-**Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(variable expression of Ley antigen in **Helicobacter pylori**: anal. of .alpha.-(1.fwdarw.2)-L-**fucosyltransferase** gene)
RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 17 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:806793 HCAPLUS
DN 130:62948
TI .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis
IN Taylor, Diane E.; Ge, Zhongming
PA The Governors of the University of Alberta, Can.
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N015-54
ICS C12N009-10; C12N015-62; C07K016-40; G01N033-573; C12Q001-68; C12P019-00; C12N009-10; C12R001-01
CC 7-2 (Enzymes)

Section cross-reference(s): 3, 9

FAN.CNT 1

	PATENT NO.	KIND	DATE		APPLICATION NO.	DATE	
PI	WO 9855630	A2	19981210		WO 1998-CA564	19980605	<--
	WO 9855630	A3	19990304				
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG						
	AU 9880050	A1	19981221		AU 1998-80050	19980605	<--
	US 6399337	B1	20020604		US 1998-92315	19980605	<--
	US 2002068347	A1	20020606		US 2000-733524	20001207	<--
	US 2002164749	A1	20021107		US 2002-120319	20020409	<--
PRAI	US 1997-48857P	P	19970606	<--			
	US 1998-92315	A3	19980605	<--			
	WO 1998-CA564	W	19980605	<--			

AB A bacterial .alpha.1,3-**fucosyltransferase** gene and deduced amino acid sequence is provided from **Helicobacter pylori**. An unusual feature of the open reading frame is the presence of 8 direct repeats of 21 nucleotides (7 amino acid repeats proximal to the C-terminus). The amino acid sequence is highly conserved except for the repeat regions. The gene is useful for prepg. .alpha.1,3-**fucosyltransferase** polypeptide, and active fragment thereof, which can be used in the prodn. of oligosaccharides such as Lewis X, Lewis Y, and **sialyl Lewis X**, which are structurally similar to certain tumor-assocd. carbohydrate antigens found in mammals. These product glycoconjugates also have research and diagnostic utility in the development of assays to detect mammalian tumors. In addn. the polypeptide of the invention can be used to develop diagnostic and research assays to det. the presence of **H. pylori** in human specimens.

ST **fucosyltransferase** gene fucT sequence Helicobacter; oligosaccharide synthesis **fucosyltransferase** Helicobacter

IT Infection
(bacterial, diagnostic of; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Diagnosis
(cancer; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Neoplasm
(diagnosis; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Gene, microbial
RL: ANT (Analyte); BPN (Biosynthetic preparation); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PREP (Preparation)
(fucT; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Oligosaccharides, preparation
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(**fucose**-contg.; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Antibodies
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)

(monoclonal; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT DNA sequences
(of .alpha.1,3-**fucosyltransferase** gene fucT of **Helicobacter pylori**)

IT Protein sequences
(of .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori**)

IT **Helicobacter pylori**
Immunoassay
Molecular cloning
Nucleic acid hybridization
PCR (polymerase chain reaction)
Plasmid vectors
Repeat motifs (protein)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Antibodies
Probes (nucleic acid)
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT Fusion proteins (chimeric proteins)
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 193834-50-3P 193837-02-4P 196223-16-2P 217793-39-0P 217793-40-3P 217793-41-4P
RL: ANT (Analyte); BPN (Biosynthetic preparation); CAT (Catalyst use); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(amino acid sequence; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 197004-40-3P
RL: ANT (Analyte); BPN (Biosynthetic preparation); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PREP (Preparation)
(nucleotide sequence; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 9023-70-5, Glutamine synthase
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(selectable marker for plasmid vectors; .alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 68247-53-0P, .alpha.1,3-**Fucosyltransferase**
RL: ANT (Analyte); BPN (Biosynthetic preparation); CAT (Catalyst use); PRP (Properties); ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 71208-06-5P, Lewis X **98603-84-0P, Sialyl-Lewis X**
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT 15839-70-0, GDP-**fucose** 73793-07-4
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

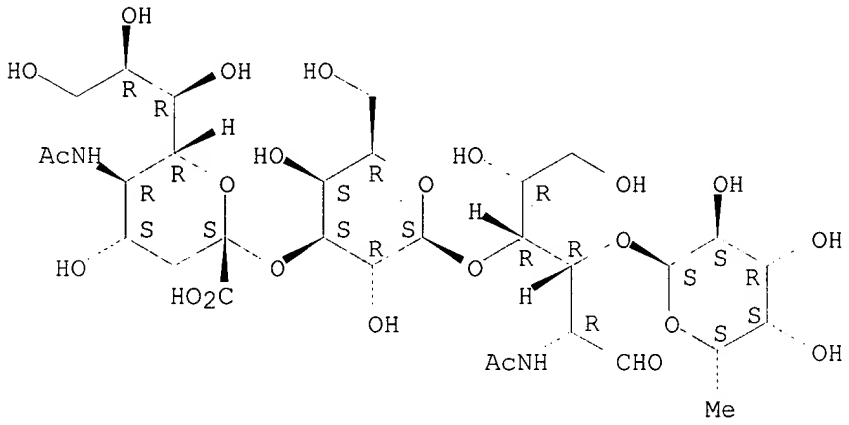
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

IT **98603-84-0P, Sialyl-Lewis X**
RL: BPN (Biosynthetic preparation); BIOL (Biological study); PREP (Preparation)
(.alpha.1,3-**fucosyltransferase** of **Helicobacter pylori** and its use for oligosaccharide synthesis)

RN 98603-84-0 HCAPLUS

CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)]-2-(acetylamino)-2-deoxy- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L117 ANSWER 18 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 1998:321795 HCAPLUS

DN 129:92671

TI Simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides. Molecular mimicry between **H. pylori** lipopolysaccharides and human gastric epithelial cell surface glycoforms

AU Monteiro, Mario A.; Chan, kenneth H. N.; Rasko, David A.; Taylor, Diane E.; Zheng, P. Y.; Appelmelk, Ben J.; Wirth, Hans-Peter; Yang, Manqiao; Blaser, Martin J.; Hynes, Sean O.; Moran, Anthony P.; Perry, Malcolm B.

CS Canadian Bacterial Diseases Network, National Research Council, Ottawa, ON, K1A 0R6, Can.

SO Journal of Biological Chemistry (**1998**), 273(19), 11533-11543
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 9

AB Previous structural investigations performed on the lipopolysaccharides (LPSs) from the human gastric pathogen **Helicobacter pylori** have revealed that these cell surface glycan mols. express type-2 partially **fucosylated**, glucosylated, or galactosylated **N-acetyllactosamine** O antigen chains (O-chains) of various lengths, which may or may not be terminated at the nonreducing end by Lewis X (Lex) and/or Ley blood group epitopes in mimicry of human cell surface glycoconjugates and glycolipids. Subsequently, serol. expts. with com. available Lewis-specific monoclonal antibodies also have recognized the presence of Lex and Ley blood group antigens in **H. pylori** but, in addn., have indicated the presence of type 1-chain Lea, Leb, and Led (H-type 1) blood group epitopes in some **H.**

pylori strains. To confirm their presence, structural studies and addnl. serol. expts. were undertaken on **H. pylori** strains suspected of carrying type-1 chain epitopes. These investigations revealed that the O-chain region of **H. pylori** strain UA948 carried both Lea (type 1) and Lex (type 2) blood group determinants. The O-chain from **H. pylori** UA955 LPS expressed the terminal Lewis disaccharide (type 1 chain) and Lex and Ley antigens (type 2). The O-chain of **H. pylori** J223 LPS carried the type 1 chain precursor Lec, the H-1 epitope (Led, type 1 chain) and an elongated **nonfucosylated** type 2 **N-acetyllactosamine** chain (i antigen). Thus, O-chains from **H. pylori** LPSs can also express **fucosylated** type 1 sequences, and the LPS from a single **H. pylori** strain may carry O-chains with type 1 and 2 Lewis blood groups simultaneously. That monoclonal antibodies putatively specific for the Leb determinant can detect glycan substructures (Le disaccharide, Lec, and Led) of Leb indicates their nonspecificity. The expression of both type 1 and 2 Lewis antigens by **H. pylori** LPSs mimics the cell surface glycomols. present in both the gastric superficial (which expresses mainly type 1 determinants) and the superficial and glandular epithelium regions (both of which express predominantly type 2 determinants). Therefore, each **H. pylori** strain may have a different niche within the gastric mucosa, and each individual LPS blood group antigen may have a dissimilar role in **H. pylori** adaptation.

ST Lewis antigen structure Helicobacter lipopolysaccharide mimicry

IT Antigens
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(I antigen; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Le**; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lea**; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**Leb**; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Antigens
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(Lec; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Antigens
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP

(Properties); BIOL (Biological study); OCCU (Occurrence)
(Led; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Ley**; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(O; simultaneous expression of type 1 and type 2 **Lewis** blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Lipopolysaccharides
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(bacterial; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Stomach
(epithelium; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Stomach, disease
(gastritis; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Antibodies
RL: ARG (Analytical reagent use); BUU (Biological use, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(monoclonal, BG-6; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Ulcer
(peptic; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT Adaptation, animal
Stomach, neoplasm
(simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT O antigen
RL: PRP (Properties)
(simultaneous expression of type 1 and type 2 Lewis blood group

antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT **Helicobacter pylori**
(strains UA948, UA955, and J223; simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT 86782-05-0
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(s simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

IT 71036-41-4 75598-07-1 79951-60-3 81243-84-7 81275-98-1 103429-56-7
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(simultaneous expression of type 1 and type 2 Lewis blood group antigens by **Helicobacter pylori** lipopolysaccharides in relation to mimicry of human gastric epithelial cell surface glycoforms)

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Amano, K; Clin Diagn Lab Immun 1997, V4, P540 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(4) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(5) Aspinall, G; Carbohydr Lett 1994, V1, P156
(6) Aspinall, G; Eur J Biochem 1997, V248, P592 HCAPLUS
(7) Aspinall, G; Ir J Med Sci 1997, V166(Suppl 3), P26
(8) Berg, D; Trends Microbiol 1997, V12, P468
(9) Blanchard, D; Rev Fr Transfus Hemobiol 1992, V35, P239 MEDLINE
(10) Ciucanu, I; Carbohydr Res 1984, V131, P209 HCAPLUS
(11) Davidson, J; Gastroenterology 1992, V103, P1552 MEDLINE
(12) Dell, A; Carbohydr Res 1990, V200, P59 HCAPLUS
(13) Dubois, M; Anal Chem 1956, V28, P350 HCAPLUS
(14) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(15) Egge, H; Mass Spectrom Rev 1987, V6, P331 HCAPLUS
(16) Ge, Z; J Biol Chem 1997, V272, P21357 HCAPLUS
(17) Henry, S; Vox Sang 1995, V69, P166 HCAPLUS
(18) Hitchcock, P; J Bacteriol 1983, V154, P269 HCAPLUS
(19) Imberty, A; Bioorg Med Chem 1996, V4, P1979 HCAPLUS
(20) Kobayashi, K; Amer J Gastroenterol 1993, V88, P919 MEDLINE
(21) Leotein, K; Carbohydr Res 1978, V62, P359
(22) Martin, S; J Biol Chem 1997, V272, P21349 HCAPLUS
(23) Mollicone, R; Lab Invest 1985, V53, P219 MEDLINE
(24) Monteiro, M; FEMS Microbiol Lett 1997, V154, P103 HCAPLUS
(25) Monteiro, M; Glycobiology 1998, V8, P107 HCAPLUS
(26) Moran, A; J Bacteriol 1997, V179, P6453 HCAPLUS
(27) Rieschel, E; Curr Top Microbiol Immunol 1996, V216, P39
(28) Sawardeker, J; Anal Chem 1967, V39, P1602
(29) Sherburne, R; Infect Immun 1995, V63, P4564 HCAPLUS
(30) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(31) Suda, Y; J Biochem (Tokyo) 1997, V121, P1129 HCAPLUS
(32) Taylor, D; Am J Clin Pathol 1987, V87, P49 MEDLINE
(33) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(34) Towbin, H; Proc Natl Acad Sci U S A 1979, V76, P4350 HCAPLUS
(35) Tsai, C; Ann Biochem 1982, V119, P115 HCAPLUS
(36) Walsh, E; Ir J Med Sci 1997, V166(Suppl 3), P27
(37) Westphal, O; Methods Carbohydr Chem 1965, V5, P83 HCAPLUS
(38) Whitfield, C; Trends Microbiol 1995, V3, P178 MEDLINE

(39) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(40) Wirth, H; Gastroenterology 1997, V112, P331
(41) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS

L117 ANSWER 19 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:7807 HCAPLUS
DN 128:190186
TI Phase variation in **Helicobacter pylori** lipopolysaccharide
AU Appelmelk, B. J.; Shiberu, B.; Trinks, C.; Tapsi, N.; Zheng, P. Y.; Verboom, T.; Maaskant, J.; Hokke, C. H.; Schiphorst, W. E. C. M.; Blanchard, D.; Simoons-Smit, I. M.; Van Den Eijnden, D. H.; Vandenbroucke-Grauls, C. M. J. E.
CS Department of Medical Microbiology, Medical School, Vrije Universiteit, Amsterdam, 1081 BT, Neth.
SO Infection and Immunity (**1998**), 66(1), 70-76
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB **Helicobacter pylori** NCTC 11637 lipopolysaccharide (LPS) expresses the human blood group antigen Lewis x (Lex) in a polymeric form. Lex is .beta.-D-galactose-(1-4)-[.alpha.-L-**fucose**-(1-3)]-.beta.-D-acetylglucosamine. Schematically the LPS structure is (Lex)n-core-lipid A. In this report, we show that Lex expression is not a stable trait but that LPS displays a high frequency (0.2 to 0.5%) of phase variation, resulting in the presence of several LPS variants in one bacterial cell population. One type of phase variation implied the loss of .alpha.1,3-linked **fucose**, resulting in variants that expressed nonsubstituted polylactosamines (also called the i antigen), i.e., Lex minus **fucose**; LPS: (lactosamine)n-core-lipid A. The switch of Lex to i antigen was reversible. A second group of variants arose by loss of polymeric main chain which resulted in expression of monomeric Ley; LPS: (Ley)-core-lipid A. A third group of variants arose by acquisition of .alpha.1,2-linked **fucose** which hence expressed Lex plus Ley; LPS: (Ley) (Lex)n-core-lipid A. The second and third group of variants switched back to the parental phenotype [(Lex)n-core-lipid A] in lower frequencies. Part of the variation can be ascribed to altered expression levels of glycosyltransferase levels as assessed by assaying the activities of galactosyl-, **fucosyl-**, and N-acetylglucosaminyltransferases. Clearly phase variation increases the heterogeneity of **H. pylori**, and this process may be involved in generating the very closely related yet genetically slightly different strains that have been isolated from one patient.
ST lipopolysaccharide phase variation Helicobacter
IT Antigens
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(I antigen; phase variation in **Helicobacter pylori** lipopolysaccharide)
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; phase variation in **Helicobacter pylori** lipopolysaccharide)
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(**Ley**; phase variation in **Helicobacter pylori** lipopolysaccharide)
IT Antigenic variation
Helicobacter pylori

(phase variation in **Helicobacter pylori** lipopolysaccharide)
IT Lipopolysaccharides
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(phase variation in **Helicobacter pylori** lipopolysaccharide)
IT 9031-68-9, Galactosyltransferase 9054-49-3, N-Acetylglucosaminyltransferase 56626-18-7, **Fucosyltransferase**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(phase variation in **Helicobacter pylori** lipopolysaccharide)

L117 ANSWER 20 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:498185 HCAPLUS
DN 127:173588
TI Chemical structures of lipopolysaccharides: a window on strain to strain variations in **Helicobacter pylori**
AU Aspinall, Gerald O.; Monteiro, Mario A.; Moran, Anthony P.
CS Department of Chemistry, York University, Toronto, ON, M3J 1P3, Can.
SO Campylobacters, Helicobacters, and Related Organisms, [Proceedings of the International Workshop on Campylobacters, Helicobacters, and Related Organisms], 8th, Winchester, UK, July 10-13, 1995 (**1996**), Meeting Date 1995, 683-686. Editor(s): Newell, Diane G.; Ketley, Julian M.; Feldman, Roger A. Publisher: Plenum, New York, N. Y.
CODEN: 64TNAY
DT Conference
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB Lipopolysaccharide samples were examd. from 4 different **H. pylori** strains. They were of considerable complexity and differed in general architecture from those of other Gram-neg. bacteria. There may be segments of variable structure which are interposed between the conserved inner core oligosaccharide and the largely repetitive O antigen chains. The repeating structure of the O chains consisted of **fucosylated** N-acetyllactosaminoglycans with Lewisx determinants, an example of mol. mimicry of human glycoconjugates in bacterial polysaccharides. The inner core oligosaccharide region, which was the same in all 4 lipopolysaccharide samples, is a phosphorylated hexasaccharide unit with a 3-deoxy-D-mannooctulosonic acid reducing unit. Other strains had lipopolysaccharides contg. the Lewisy determinant and intervening regions contg. D-glycero-D-mannoheptose.
ST lipopolysaccharide Helicobacter
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**Ley**; in lipopolysaccharides of **Helicobacter pylori**)
IT **Helicobacter pylori**
(strain variations in chem. structures of lipopolysaccharides of **Helicobacter pylori**)
IT Lipopolysaccharides
O antigen
RL: PRP (Properties)
(strain variations in chem. structures of lipopolysaccharides of **Helicobacter pylori**)
IT 1961-73-5, D-glycero-D-manno-Heptose 71208-06-5, Lewis x
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(in lipopolysaccharides of **Helicobacter pylori**)

L117 ANSWER 21 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 1997:315719 HCAPLUS
DN 127:3804
TI Transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as a model for Helicobacter infection
IN Falk, Per; Gordon, Jeffrey I.
PA Washington University, USA
SO U.S., 24 pp.
CODEN: USXXAM
DT Patent
LA English
IC ICM C12N005-00
ICS A61K049-00; G01N033-567
NCL 800002000
CC 14-3 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 3, 10
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5625124	A	19970429	US 1994-273411	19940711 <--
PRAI US 1994-273411		19940711 <--		

AB Transgenic non-human animals expressing human genes for enzymes involved in the formation of **fucosylated** epitopes bound by **Helicobacter pylori** are described for use as a model for **H. pylori** infection. These animals can be used to study the development of infection, screen for inhibitors of infection, and to study the effect of dietary, environmental and physiol. change on the course of the disease. Cells of the gut epithelium of these animals present one or more surface antigens that act as receptors for the bacterium **H. pylori**, a known causative agent of acid peptic disease, such as gastritis, stomach ulcers, duodenal ulcers, and strongly correlated with the development of gastric neoplasia. The genes for human GDP-L-**fucose**: .beta.-D-galactoside-2-.alpha.-L-**fucosyltransferase** and GDP-L-**fucose**: .beta.-D-N-Acetylglucosaminide 3,4-.alpha.-L-**fucosyltransferase** are used and are expressed from the Fabpl promoter to direct digestive tract-specific expression of the genes. Methods for making and using the transgenic animals are also disclosed. The transgenic animals can be used to screen for compds. and conditions which block binding of **H. pylori** to the gut epithelium and/or ameliorate the **H. pylori**-assocd. pathogenesis of acid peptic disease and gastric adenocarcinoma.

ST Helicobacter infection animal model **fucosyltransferase** gene; H antigen Helicobacter infection animal model; Lewis antigen Helicobacter infection animal model

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
(ABH, H-1, presentation on animal gut epithelium of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Fabpl, gut-specific expression of genes from promoter of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT Promoter (genetic element)
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(Fabpl, gut-specific expression of genes from; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter**

pylori as model for Helicobacter infection)
IT Gene, animal
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(H, expression in transgenic animals of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Plasmid vectors
(LF.alpha.1.2Fuc, **fucosyltransferase** gene on, expression in transgenic mice of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Plasmid vectors
(LF.alpha.1.3/4Fuc, **fucosyltransferase** gene on, expression in transgenic mice of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Gene, animal
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Le, expression in transgenic animals of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
(**Leb**, presentation on animal gut epithelium of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Digestive tract
(epithelium, presentation of **fucosyl** polysaccharides on surface of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT cDNA sequences
(for **fucosyltransferases** of human; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Polysaccharides, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**fucosylated**, as ligands for **Helicobacter pylori**; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Adhesion, biological
(of Helicobacter to gut epithelium, identification of inhibitors of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT Protein sequences
(of **fucosyltransferases** of human; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT **Helicobacter pylori**
(transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)
IT 131198-88-4 131361-39-2
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; transgenic animals presenting **fucosylated**

epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT 37277-69-3, Lewis **fucosyltransferase 56093-23-3**, e.c. 2.4.1.69
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human gene for, expression in transgenic animals of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT 190086-76-1
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT 138186-21-7 140030-38-2
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

IT **56093-23-3**, e.c. 2.4.1.69
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(human gene for, expression in transgenic animals of; transgenic animals presenting **fucosylated** epitopes bound by **Helicobacter pylori** as model for Helicobacter infection)

RN 56093-23-3 HCAPLUS
CN Fucosyltransferase, guanosine diphosphofucose-galactoside 2-L- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L117 ANSWER 22 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:79335 HCAPLUS
DN 124:111908
TI Lipopolysaccharides of **Helicobacter pylori** strains P466 and MO19: structures of the O antigen and core oligosaccharide regions
AU Aspinall, Gerald O.; Monteiro, Mario A.
CS Department of Chemistry, York University, North York, ON, M3J 1P3, Can.
SO Biochemistry (**1996**), 35(7), 2498-504
CODEN: BICHAW; ISSN: 0006-2960
PB American Chemical Society
DT Journal
LA English
CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
AB Lipopolysaccharides (LPS) from PhOH-H2O extn. of dyspeptic (P466) and asymptomatic (MO19) strains of **H. pylori** were each isolated as water-sol. material of high relative mol. mass (high Mr) and as water-insol. gels of low Mr. Chem. and spectroscopic analyses of the sol. LPS and oligosaccharides liberated from the water-insol. gels led to proposed structures for chains comprising the O antigen, intervening, and core regions. As in the LPS from the type strain NCTC 11637, the O antigen region is characterized by the presence of extended chains with **fucosylated** and **nonfucosylated N-acetyllactosamine** units, the former carrying .alpha.-L-**fucopyranose** units at O-3 of .beta.-D-GlcNAc residues. The structure of the P466 LPS differs from that of the type strain in termination of the O chain by a Lewisy (Ley) antigenic determinant

[.alpha.-L-Fuc(1.fwdarw.2).beta.-D-Gal(1.fwdarw.4)[.alpha.-L-Fuc(1.fwdarw.3)].beta.-D-GlcNAc] but also has internal Lewisx (Lex) units. The inner core region of the P466 LPS is indistinguishable from that in the type strain. In contrast, the O antigen region of the LPS from strain MO19 consists of a single Ley epitope linked via a 3-linked .beta.-D-Gal to an intervening region on the basis of a sequence of 3-linked D-glycero-.alpha.-D-mannoheptose residues which is in turn linked to an inner core identical to that in the type strain and the P466 strain. LPS from the 3 **H. pylori** strains display mol. mimicry of human cell surface glycoconjugates but may vary in the expression of Lex or Ley determinants, the degree of O antigen chain extension, or in the presence of an addnl. region between the inner core and the O antigen.

ST O antigen Helicobacter lipopolysaccharide structure; oligosaccharide Helicobacter lipopolysaccharide structure

IT **Campylobacter pyloridis**
(structures of the O antigen and core oligosaccharide regions of lipopolysaccharides of **Helicobacter pylori** strains P466 and MO19)

IT Lipopolysaccharides
Oligosaccharides
RL: PRP (Properties)
(structures of the O antigen and core oligosaccharide regions of lipopolysaccharides of **Helicobacter pylori** strains P466 and MO19)

IT Antigens
RL: PRP (Properties)
(O, structures of the O antigen and core oligosaccharide regions of lipopolysaccharides of **Helicobacter pylori** strains P466 and MO19)

L117 ANSWER 23 OF 24 HCAPLUS COPYRIGHT 2003 ACS

AN 1996:79334 HCAPLUS

DN 124:111907

TI Lipopolysaccharide of the **Helicobacter pylori** type strain NCTC 11637 (ATCC 43504): structure of the O antigen chain and core oligosaccharide regions

AU Aspinall, Gerald O.; Monteiro, Mario A.; Pang, Henrianna; Walsh, Evelyn J.; Moran, Anthony P.

CS Department of Chemistry, York University, North York, ON, M3J 1P3, Can.

SO Biochemistry (**1996**), 35(7), 2489-97
CODEN: BICHAW; ISSN: 0006-2960

PB American Chemical Society

DT Journal

LA English

CC 10-1 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 33

AB Smooth- and rough-form lipopolysaccharides from phenol-water extn. of cells from **Helicobacter pylori** type strain NCTC 11637 were isolated as the water-sol. component of high-Mr and water-insol. low-Mr gel. Structural investigations were performed on the intact water-sol. smooth-form lipopolysaccharide, various oligosaccharides formed as chem. and enzymic degrdn. products, and three oligosaccharide fractions liberated by acetic acid hydrolysis from the water-insol. rough-form lipopolysaccharide. A structure is proposed for the complete polysaccharide component of the smooth-form lipopolysaccharide comprising the O antigen chain, an intervening region, and the inner core oligosaccharide on the basis of 1H and 13C NMR expts., fast-atom bombardment/mass spectrometry, and methylation linkage anal. of permethylated oligo- and polysaccharide derivs. The most striking feature of the O antigen region in the lipopolysaccharide is the presence of extended chains with **fucosylated** and **nonfucosylated** **N-acetyllactosamine** (LacNAc) units that mimic human cell surface glycoconjugates in normal human granulocytes. The chains are

terminated by di- or trimeric Lewisx (Lex) determinants, which are also found in tumor-assocd. carbohydrate antigens in many adenocarcinomas.
ST Helicobacter lipopolysaccharide antigen core oligosaccharide structure; antigen O structure lipopolysaccharide Helicobacter
IT **Campylobacter pyloridis**
(structure of the O antigen chain and core oligosaccharide regions of lipopolysaccharide of the **Helicobacter pylori** type strain NCTC 11637)
IT Lipopolysaccharides
Oligosaccharides
RL: PRP (Properties)
(structure of the O antigen chain and core oligosaccharide regions of lipopolysaccharide of the **Helicobacter pylori** type strain NCTC 11637)
IT Antigens
RL: PRP (Properties)
(O, structure of the O antigen chain and core oligosaccharide regions of lipopolysaccharide of the **Helicobacter pylori** type strain NCTC 11637)

L117 ANSWER 24 OF 24 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:411777 HCAPLUS
DN 122:184434
TI Expression of a human .alpha.-1,3/4-**fucosyltransferase** in the pit cell lineage of FVB/N mouse stomach results in production of Leb-containing glycoconjugates: a potential transgenic mouse model for studying **Helicobacter pylori** infection
AU Falk, Per G.; Bry, Lynn; Holgersson, Jan; Gordon, Jeffrey I.
CS Sch. Med., Washington Univ., St. Louis, MO, 63110, USA
SO Proceedings of the National Academy of Sciences of the United States of America (**1995**), 92(5), 1515-19
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
CC 14-3 (Mammalian Pathological Biochemistry)
AB **Helicobacter pylori** is a human pathogen assocd. with the development of gastric and duodenal ulcers and gastric adenocarcinoma. To test the hypothesis that the human Lewisb blood group antigen (Leb) functions as a receptor for the bacteria's adhesins and mediates its attachment to gastric pit and surface mucous cells, a human .alpha.-1,3/4-**fucosyltransferase** was expressed in these cell lineages in FVB/N transgenic mice. The **fucosyltransferase** directed prodn. of the Leb epitope without any apparent effect on the proliferation and differentiation programs of this lineage. Moreover, clin. isolates of **H. pylori** bound to these cells in transgenic mice but not in their normal littermates. Binding was blocked by pretreatment of the bacteria with sol. Leb. This mouse model could be useful for examg. the mol. pathogenesis of diseases caused by **H. pylori** infection. Creating novel pathways for prodn. of specific oligosaccharides in selected cell lineages of transgenic animals represents an approach for examg. the role of complex carbohydrates in regulating cellular differentiation and host-microbe interactions.
ST **fucosyltransferase** stomach Lewis antigen Helicobacter infection; transgenic mouse **fucosyltransferase** Helicobacter infection mouse
IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Leb-contg. glycoconjugates are **Helicobacter pylori** receptors in stomach)
IT **Campylobacter pyloridis**
Mouse
Transformation, genetic

(transgenic mouse model for studying **Helicobacter pylori fucosyltransferase**-mediated formation of human Leb-contg. glycoconjugates in stomach)

IT **Blood-group substances**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**Leb**, transgenic mouse model for studying **Helicobacter pylori fucosyltransferase** -mediated formation of human **Leb**-contg. glycoconjugates in stomach)

IT Adhesion
(bio-, **Helicobacter pylori fucosyltransferase**-mediated formation of human Leb-contg. glycoconjugates mediates **H. pylori** adhesion to stomach cells)

IT 37277-69-3, .alpha.-1,3/4-**Fucosyltransferase**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(transgenic mouse model for studying **Helicobacter pylori fucosyltransferase**-mediated formation of human Leb-contg. glycoconjugates in stomach)

=> d all hitstr tot 12-14,16-19,21-23

L125 ANSWER 1 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2001:452866 HCAPLUS
DN 135:71250
TI Novel **Helicobacter pylori**-binding substances and use thereof
IN Karlsson, Karl-anders; Leonardsson, Irene; Teneberg, Susann; Angstroem, Jonas
PA A+ Science Invest AB, Swed.
SO PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-702
ICS A61P001-04; A61P031-04
CC 1-5 (**Pharmacology**)
Section cross-reference(s): 10, 15, 17, 33, **63**
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001043751	A1	20010621	WO 2000-SE2567	20001215 <--
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1237558	A1	20020911	EP 2000-987920	20001215 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
NO 2002002890	A	20020815	NO 2002-2890	20020617 <--
PRAI SE 1999-4581	A	19991215 <--		
WO 2000-SE2567	W	20001215		

OS MARPAT 135:71250

AB **Helicobacter pylori**-binding substances comprising Gal.beta.3GlcNAc or Gal.beta.3GalNAc are described, as well as use thereof in pharmaceutical compns. and food-stuff, and methods for treatment of conditions due to the presence of **Helicobacter pylori**. Also use of said substance for the identification of bacterial adhesions, for the prodn. of a vaccine against **Helicobacter pylori** , for diagnosis of **Helicobacter pylori** infections, for typing of **Helicobacter pylori**, for identification of **Helicobacter pylori** binding substances and for inhibition of the binding of **Helicobacter pylori** is described.

ST Helicobacter binding substance glycosphingolipid

IT Micelles
(**Helicobacter pylori**-binding compds. in; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Structure-activity relationship
(**Helicobacter pylori**-binding; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Stomach, neoplasm
(adenocarcinoma, inhibitors; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Infection
(bacterial, with **Helicobacter pylori**, diagnosis of; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Drug delivery systems
(carriers; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Stomach, disease
(chronic gastritis; novel **Helicobacter pylori** -binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Antibiotics
(conjugates with **Helicobacter pylori**-binding compds.; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Polysaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with **Helicobacter pylori**-binding compds.; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Digestive tract
(disease; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Intestine, disease
(duodenum, ulcer; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Antitumor agents
(gastric adenocarcinoma; novel **Helicobacter pylori**

-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Glycosphingolipids
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(globosides; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Milk substitutes
(human; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Adhesins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(identification of bacterial; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Glycoproteins, specific or class
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neoglycoproteins; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Antitumor agents
(non-Hodgkin's lymphoma; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Antibacterial agents
Antiulcer agents
Drug delivery systems
Drug screening
Food
Food additives
Helicobacter pylori
Molecular modeling
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Cerebrosides
Glycolipids
Glycoproteins, general, biological studies
Glycosphingolipids
Oligosaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Diagnosis
(of **Helicobacter pylori** infections; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Genotyping (method)
(of **Helicobacter pylori**; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Death
(sudden infant death syndrome, treatment; novel **Helicobacter**

pylori-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Vaccines
(to **Helicobacter pylori**, prodn. of; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Heart, disease
Liver, disease
(treatment; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT Stomach, disease
(ulcer; novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT 14116-68-8
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); RCT (Reactant); **THU (Therapeutic use)**; BIOL (Biological study); RACT (Reactant or reagent); USES (Uses)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT 345305-75-1P 345305-76-2P
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT 3554-90-3 3554-90-3D, analogs **4682-48-8**, Lactosylceramide **11034-93-8** 13007-32-4 **35960-33-9**, Gangliotriaosylceramide 50787-09-2 50787-09-2D, analogs **56573-54-7**, Neolactotetraosylceramide **71012-19-6**, Gangliotetraosylceramide **71950-33-9**, Lactotetraosylceramide **71965-57-6**, Globotriaosylceramide **73201-40-8** **73467-80-8**, Lactotriaosylceramide 75660-79-6, Globotetraose **77538-29-5 77538-32-0 77538-33-1** **87501-61-9 88161-63-1 89678-48-8** **89678-50-2 91847-19-7** 100787-31-3D, Polylactosamine, conjugates with **Helicobacter pylori**-binding compds. **103842-51-9 162731-01-3 222540-55-8**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

IT 34620-78-5, Maltoheptaose 79098-13-8, 4-Hexadecylaniline
RL: RCT (Reactant); RACT (Reactant or reagent)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

(1) Ahmad, R; US 4957741 A 1990 HCAPLUS
(2) Eric, H; Archives of Biochemistry and Biophysics 1990, V277(1), P181
(3) Gold, B; Infection and Immunity 1993, V61(6), P2632 HCAPLUS
(4) Karlsson; EP 0133170 A2 1985 HCAPLUS
(5) Leffler; WO 8103175 A1 1981 HCAPLUS
(6) Mario, A; The Journal of Biological Chemistry 1998, V273(19), P11533

(7) Masayuki, M; Cancer Research 1989, V49, P5689
(8) Meike, B; Am J Clin Nutr 2000, V71, P1589
(9) Miller-Podraza, H; Infection and Immunity 1997, V65(6), P2480 HCAPLUS
(10) Murakami, M; JP 10-45602 A 1998 HCAPLUS
(11) Per, F; J Biochem 1990, V108, P466
(12) Thomas, B; Science 1993, V262(5141), P1892

IT **4682-48-8**, Lactosylceramide **11034-93-8**
35960-33-9, Gangliotriaosylceramide **56573-54-7**,
Neolactotetraosylceramide **71012-19-6**, Gangliotetraosylceramide
71950-33-9, Lactotetraosylceramide **71965-57-6**,
Globotriaosylceramide **73201-40-8 73467-80-8**,
Lactotriaosylceramide **77538-29-5 77538-32-0**
77538-33-1 87501-61-9 88161-63-1
89678-48-8 89678-50-2 91847-19-7
103842-51-9 162731-01-3 222540-55-8
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); FFD (Food or feed use); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(novel **Helicobacter pylori**-binding substances and use thereof for treatment of diseases of gastrointestinal tract and for food use)

RN 4682-48-8 HCAPLUS
CN Ceramide, 1-O-(4-O-.beta.-D-galactopyranosyl-.beta.-D-glucopyranosyl)- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 11034-93-8 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-.alpha.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 56573-54-7 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71950-33-9 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71965-57-6 HCAPLUS
CN Ceramide, 1-O-(O-.alpha.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl)- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 73201-40-8 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 73467-80-8 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 77538-29-5 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-[6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 77538-32-0 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 77538-33-1 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)-O-[.beta.-D-galactopyranosyl-(1.fwdarw.3)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 87501-61-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.alpha.-D-galactopyranosyl-(1.fwdarw.3)-O-[6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 88161-63-1 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.3)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 89678-48-8 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-[.alpha.-D-galactopyranosyl-(1.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 89678-50-2 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 91847-19-7 HCAPLUS
CN Ceramide, 1-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.4)-O-[O-6-deoxy-.alpha.-L-galactopyranosyl-(1.fwdarw.2)-O-[.alpha.-D-galactopyranosyl-(1.fwdarw.3)]-.beta.-D-galactopyranosyl-(1.fwdarw.3)]-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 103842-51-9 HCAPLUS
CN Ceramide, 1-O-[O-[N-(hydroxyacetyl)-.alpha.-neuraminosyl]-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 162731-01-3 HCAPLUS
CN Ceramide, 1-O-[O-.alpha.-D-galactopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 222540-55-8 HCAPLUS
CN Ceramide, 1-O-(O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-amino-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl)- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 2 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:862195 HCAPLUS
DN 135:44775
TI **Helicobacter pylori** infection and gastrointestinal immunity
AU Sugiyama, Toshiro; Asaka, Masahiro
CS Graduate School, Hokkaido University, Japan
SO G.I. Research (**2000**), 8(5), 372-378
CODEN: GIREFM; ISSN: 0918-9408
PB Sentan Igakusha
DT Journal; General Review
LA Japanese
CC 15-0 (Immunochemistry)
Section cross-reference(s): 14
AB A review with 16 refs. discussing gastrointestinal immune responses to **Helicobacter pylori**. Topics discussed include gastric mucosal immunity, mucosal antibody prodn., roles of T lymphocytes, cytokines, MHC class II antigens, **H. pylori** antigens, and antigenic mimicry between **H. pylori** lipopolysaccharide and host Lewis blood group antigens. Immune evasion mechanism is also discussed.
ST review gastrointestinal immunity Helicobacter cytokine antigen
IT **Blood-group substances**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(**Le**; gastrointestinal immune responses to **H. pylori** infection in relation to)
IT Histocompatibility antigens
RL: **BAC (Biological activity or effector, except adverse)**; BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(MHC (major histocompatibility complex), class II; gastrointestinal immune responses to **H. pylori** infection)

IT Lipopolysaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(bacterial; gastrointestinal immune responses to **H. pylori** infection in relation to)

IT CD4-positive T cell
CD8-positive T cell
Helicobacter pylori
(gastrointestinal immune responses to **H. pylori** infection)

IT Antibodies
Cytokines
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(gastrointestinal immune responses to **H. pylori** infection)

IT Stomach
(mucosa; gastrointestinal immune responses to **H. pylori** infection)

L125 ANSWER 3 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:202305 HCAPLUS
DN 133:86569
TI Functional genomics of **Helicobacter pylori**: identification of a .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide
AU Logan, S. M.; Conlan, J. W.; Monteiro, M. A.; Wakarchuk, W. W.; Altman, E.
CS Institute for Biological Sciences, National Research Council of Canada, Ottawa, ON, K1A OR6, Can.
SO Molecular Microbiology (**2000**), 35(5), 1156-1167
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
CC 10-2 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 7
AB A previously annotated open reading frame (ORF) (HP0826) from **Helicobacter pylori** was cloned and expressed in Escherichia coli cells and detd. to be a .beta.-1,4-galactosyltransferase that used GlcNAc as an acceptor. Mutational anal. in **H. pylori** strains demonstrated that this enzyme plays a key role in the biosynthesis of the type 2 **N-acetyllactosamine** (LacNAc) polysaccharide O-chain backbone, by catalyzing the addn. of Gal to GlcNAc. To examine the potential role of this O-chain structure in bacterial colonization of the host stomach, the mutation was introduced into **H. pylori** strain SS1 which is known to be capable of colonizing the gastric mucosa of mice. Compared with the parental strain, mutated SS1 was less efficient at colonizing the murine stomach.
ST galactosyltransferase lipopolysaccharide formation Helicobacter
IT Gene, microbial
RL: **BAC (Biological activity or effector, except adverse)**; BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(HP0826; **Helicobacter pylori** .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide)

IT **Helicobacter pylori**
(**Helicobacter pylori** .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide)

IT Lipopolysaccharides

RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(**Helicobacter pylori** .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide)

IT 9054-94-8, Acetylglucosamine .beta.-1,4-galactosyltransferase
RL: **BAC (Biological activity or effector, except adverse)**; BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**Helicobacter pylori** .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide)

IT 193838-64-1, Galactosyltransferase, uridine diphosphogalactose-acetylglucosamine (**Helicobacter pylori** strain 26695 gene HP0826)
RL: **BAC (Biological activity or effector, except adverse)**; BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(nucleotide sequence; **Helicobacter pylori** .beta.-1,4 galactosyltransferase and generation of mutants with altered lipopolysaccharide)

RE.CNT 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(2) Aspinall, G; Biochemistry 1996, V35, P2489 HCAPLUS
(3) Aspinall, G; Biochemistry 1996, V35, P2498 HCAPLUS
(4) Aspinall, G; Eur J Biochem 1997, V248, P592 HCAPLUS
(5) Blanchard, T; Cell Immunol 1999, V191, P74 HCAPLUS
(6) Blaser, M; Gastroenterology 1992, V102, P720 MEDLINE
(7) Boren, T; Science 1993, V262, P1892 HCAPLUS
(8) Chan, N; Glycobiology 1995, V5, P683 HCAPLUS
(9) Ciucanu, I; Carbohydr Res 1984, V131, P209 HCAPLUS
(10) Conlan, J; Can J Microbiol 1999, V45, P975 HCAPLUS
(11) Cope, L; Mol Microbiol 1994, V5, P1113
(12) Dell, A; Carbohydr Res 1990, V200, P59 HCAPLUS
(13) Dubois, M; Anal Chem 1956, V28, P350 HCAPLUS
(14) Eaton, K; Infect Immun 1991, V59, P2470 HCAPLUS
(15) Engvall, E; J Immunol 1972, V109, P129 HCAPLUS
(16) Ermak, T; J Exp Med 1998, V188, P2277 HCAPLUS
(17) Evans, D; J Bacteriol 1993, V175, P674 HCAPLUS
(18) Ferrero, R; Infect Immun 1998, V66, P1349 HCAPLUS
(19) Gilbert, M; Eur J Biochem 1997, V249, P187 HCAPLUS
(20) Haas, R; Mol Microbiol 1993, V8, P753 HCAPLUS
(21) Heinrichs, D; Mol Microbiol 1998, V30, P221 HCAPLUS
(22) Higgins, D; Gene 1988, V73, P237 HCAPLUS
(23) High, N; Mol Microbiol 1993, V9, P1275 HCAPLUS
(24) Inzana, T; Infect Immun 1997, V65, P4675 HCAPLUS
(25) Jarosik, G; Infect Immun 1994, V62, P4861 HCAPLUS
(26) Jennings, M; Mol Microbiol 1995, V18, P729 HCAPLUS
(27) Kwon, D; Curr Microbiol 1998, V37, P144 HCAPLUS
(28) Labigne-Roussel, A; J Bacteriol 1988, V170, P1704 HCAPLUS
(29) Laemmli, U; Nature 1970, V227, P680 HCAPLUS
(30) Lee, A; Gastroenterology 1997, V112, P1386 MEDLINE
(31) Logan, S; Infect Immun 1984, V45, P210 HCAPLUS
(32) McGowan, C; Mol Microbiol 1998, V30, P19 HCAPLUS
(33) Michetti, P; Infect Immun 1992, V60, P1786 HCAPLUS
(34) Monteiro, M; Eur J Biochem 2000, V167, P305
(35) Monteiro, M; J Biol Chem 1998, V273, P11533 HCAPLUS
(36) Moran, A; Aliment Pharmacol Ther 1996, V10(Suppl), P39
(37) Moran, A; FEMS Immunol Med Microbiol 1996, V16, P105 HCAPLUS
(38) Moran, A; FEMS Microbiol Immunol 1995, V10, P271 HCAPLUS
(39) Odenbreit, S; Gut 1995, V37(Suppl 1), PA1
(40) Pappo, J; Infect Immun 1999, V67, P337 HCAPLUS

(41) Parsonnet, J; N Engl J Med 1991, V325, P1127 MEDLINE
(42) Phadnis, S; Infect Immun 1994, V62, P1557 HCAPLUS
(43) Potter, M; FEMS Microbiol Lett 1995, V129, P75 HCAPLUS
(44) Roth, K; J Immunol 1999, V163, P1490 HCAPLUS
(45) Sambrook, J; Molecular Cloning: A Laboratory Manual 2nd ed 1989
(46) Sawardeker, J; Anal Chem 1967, V39, P1602
(47) Shirai, M; J Infect Dis 1997, V177, P72
(48) Tomb, J; Nature 1997, V388, P539 HCAPLUS
(49) Tsai, C; Anal Biochem 1982, V119, P115 HCAPLUS
(50) Tummuru, M; Mol Microbiol 1995, V18, P867 HCAPLUS
(51) Wakarchuk, W; J Biol Chem 1996, V271, P19166 HCAPLUS
(52) Wang, G; Mol Microbiol 1999, V31, P1265 HCAPLUS
(53) Westphal, O; Meth Carbohydr Chem 1965, V5, P83 HCAPLUS
(54) Whitfield, C; Mol Microbiol 1997, V23, P629 HCAPLUS
(55) Winner, L; Infect Immun 1991, V59, P977 HCAPLUS
(56) Yokota, S; Infect Immun 1998, V66, P3006 HCAPLUS
(57) Young, G; Ailment Pharmacol Ther 1992, V6, P169 HCAPLUS

L125 ANSWER 4 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 2000:81796 HCAPLUS
DN 132:221055
TI Relationship of blood group determinants on **Helicobacter pylori** lipopolysaccharide with host Lewis phenotype and inflammatory response
AU Heneghan, Michael A.; McCarthy, Ciaran F.; Moran, Anthony P.
CS Department of Medicine, Clinical Science Institute, University College Hospital Galway, National University of Ireland, Galway, Ire.
SO Infection and Immunity (**2000**), 68(2), 937-941
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 15-2 (Immunochemistry)
Section cross-reference(s): 14
AB As Lewis a (Lea) and Lewis b (Leb) blood group antigens are isoforms of Lewis x (Lex) and Lewis y (Ley) and are expressed in the gastric mucosa, we evaluated whether the patterns of expression of Lex and Ley on **Helicobacter pylori** lipopolysaccharides reflected those of host expression of Lea and Leb. When 79 patients (secretors and nonsecretors) were examd. for concordance between bacterial and host Le expression, no assocn. was found, nor was there a significant difference between the amt. of Lex or Ley expressed on isolates from ulcer and chronic gastritis patients. Also, the effect of host and bacterial expression of Le antigens on bacterial colonization and the obsd. inflammatory response was assessed. In ulcer patients, Lex expression was significantly related to neutrophil infiltration, whereas in chronic gastritis patients significant relationships were found between Lex expression and **H. pylori** colonization d., neutrophil infiltrate, and lymphocyte infiltrate. Furthermore, bacterial Ley expression was related to neutrophil and lymphocyte infiltrates. Thus, although no evidence of concordance was found between bacterial and host expression of Le determinants, these antigens may be crucial for bacterial colonization, and the ensuing inflammatory response appears, at least in part, to be influenced by Le antigens.
ST Helicobacter Lewis blood group antigen leukocyte infiltration
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; host **Lewis** phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)

(**Ley**; host **Lewis** phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT Lipopolysaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(bacterial; host Lewis phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT **Helicobacter pylori**
(host Lewis phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT Neutrophil
(infiltration; host Lewis phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT Lymphocyte
(migration; host Lewis phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
IT Stomach
(mucosa; host Lewis phenotype and inflammatory response to **H. pylori** lipopolysaccharide)
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Appelmelk, B; Immunol Today 1998, V19, P296 HCAPLUS
(2) Appelmelk, B; Infect Immun 1996, V64, P2031 HCAPLUS
(3) Dickey, W; Gut 1993, V34, P351 MEDLINE
(4) Dunn, B; Clin Microbiol Rev 1997, V10, P720 HCAPLUS
(5) Eidt, S; Helicobacter pylori gastritis and peptic ulcer 1990, P228
(6) Green, C; FEMS Microbiol Immunol 1989, V47, P321
(7) Guruge, J; Proc Natl Acad Sci USA 1998, V95, P3925 HCAPLUS
(8) Heneghan, M; FEMS Immunol Med Microbiol 1998, V20, P257 HCAPLUS
(9) Hitchcock, P; J Bacteriol 1983, V154, P269 HCAPLUS
(10) Klaamas, K; Eur J Gastroenterol Hepatol 1997, V9, P367 MEDLINE
(11) Lloyd, K; Am J Clin Pathol 1987, V87, P129 HCAPLUS
(12) Marshall, D; FEMS Immunol Med Microbiol 1999, V24, P79 HCAPLUS
(13) Mentis, A; Epidemiol Infect 1991, V106, P221 MEDLINE
(14) Mollicone, R; Lab Investig 1985, V53, P219 MEDLINE
(15) Moran, A; FEMS Immunol Med Microbiol 1995, V10, P271 HCAPLUS
(16) Moran, A; J Endotoxin Res 1996, V3, P521 HCAPLUS
(17) Moran, A; Scand J Gastroenterol 1996, V31(Suppl 215), P22
(18) O'Croinin, T; Gastroenterology 1998, V114, P690
(19) Saadi, A; Epidemiol Infect 1993, V110, P507 MEDLINE
(20) Sakamoto, J; Cancer Res 1989, V49, P745 HCAPLUS
(21) Simoons-Smit, I; J Clin Microbiol 1996, V34, P2196 MEDLINE
(22) Taylor, D; Gastroenterology 1998, V115, P1113 MEDLINE
(23) Towbin, H; Proc Natl Acad Sci USA 1979, V76, P4350 HCAPLUS
(24) Walsh, E; J Appl Microbiol 1997, V83, P67 HCAPLUS
(25) Wirth, H; Gastroenterology 1997, V113, P1091 MEDLINE
(26) Wirth, H; Infect Immun 1996, V64, P4598 HCAPLUS

L125 ANSWER 5 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:736498 HCAPLUS
DN 131:335799
TI Immunomodulatory activity of B subunits of cholera toxin, verotoxin, and heat-labile enterotoxin
IN Hirst, Timothy Raymond; Williams, Neil Andrew
PA University of Bristol, UK
SO PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K039-00
CC 15-2 (Immunochemistry)
Section cross-reference(s): **1**, 14
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9958145	A2	19991118	WO 1999-GB1461	19990510	<--
	WO 9958145	A3	20000203			
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
	AU 9939394	A1	19991129	AU 1999-39394	19990510	<--
	BR 9910305	A	20010109	BR 1999-10305	19990510	<--
	EP 1075274	A2	20010214	EP 1999-922284	19990510	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
	GB 2353472	A1	20010228	GB 2000-27072	19990510	<--
	JP 2002514607	T2	20020521	JP 2000-547996	19990510	<--
	NO 2000005599	A	20010108	NO 2000-5599	20001106	<--
PRAI	GB 1998-9958	A	19980508	<--		
	GB 1998-11954	A	19980603	<--		
	GB 1998-12316	A	19980608	<--		
	WO 1999-GB1461	W	19990510	<--		

AB The authors disclose the use of: (i) heat-labile enterotoxin B subunit (EtxB), cholera toxin B subunit (CtxB) or verotoxin B subunit (VtxB) in vaccine prepns. to alter the immune response to pathogens. In one example, the secretory IgA response to herpes virus glycoproteins is enhanced by the adjuvant activity of EtxB. In addn., the authors disclose the use of agents other than EtxB or CtxB, which have ganglioside GM1-binding activity, or an agent other than VtxB which has globotriosylceramide (Gb3)-binding activity for affecting intracellular signaling events.

ST toxin immunomodulator vaccine infection

IT Immunomodulators
(B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin)

IT Antigen presentation
(B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin for prolongation of)

IT Antigen-presenting cell
(B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin for prolongation of presentation function of)

IT Bacillus cereus
Campylobacter jejuni
Chlamydia trachomatis
Cytomegalovirus
Escherichia coli
Helicobacter pylori
Hepatitis A virus
Hepatitis B virus
Hepatitis C virus
Hepatitis delta virus
Human herpesvirus 1
Human herpesvirus 2
Human herpesvirus 3
Human herpesvirus 4
Human herpesvirus 6
Human herpesvirus 7
Human herpesvirus 8
Human immunodeficiency virus 1
Human immunodeficiency virus 2
Human parainfluenza virus

Infection
Influenza virus
Legionella pneumophila
Leishmania donovani
Malaria
Meningitis
Mycobacterium tuberculosis
Neisseria gonorrhoeae
Neisseria meningitidis
Onchocerca
Parasite
Pneumonia
Respiratory syncytial virus
Rotavirus
Salmonella enteritidis
Salmonella typhi
Sexually transmitted diseases
Staphylococcus aureus
Streptococcus mutans
Streptococcus pneumoniae
Streptococcus pyogenes
Toxoplasma gondii
Trypanosoma
Vibrio cholerae
(B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin in vaccines against)

IT Histocompatibility antigens
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(MHC (major histocompatibility complex), class I; vesicular internalization of antigen-toxin B subunit conjugates in antigen-presenting cells for enhancing presentation function of)

IT Toxins
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(Shiga-like toxin, B subunit; immunomodulatory activity of)

IT Crosslinking agents
(bifunctional; for conjugation of antigenic determinants with B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin)

IT Protein receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(cholera toxin; immunomodulators with signaling activity mediated via binding to)

IT Toxins
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(cholera, B subunit; immunomodulatory activity of)

IT Antigens
RL: **BAC (Biological activity or effector, except adverse)**; BPR (Biological process); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(conjugates, with B subunit of toxins; vesicular internalization in antigen-presenting cells of)

IT Toxins
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(enterotoxins, heat-labile, B subunit; immunomodulatory activity of)

IT Immunostimulation

(humoral; by B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin)
IT Immunity
(immunol. memory; B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin for prolongation of)
IT Vaccines
(immunomodulatory activity of B subunits of cholera toxin, verotoxin, and heat-labile enterotoxin in)
IT Signal transduction, biological
(induced by B subunits of toxins binding to gangliosides)
IT Digestive tract
Respiratory tract
(infection; B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin in vaccines against)
IT Immunity
(mucosal; B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin up-regulate antibody response in)
IT Epitopes
(of infectious agents in vaccines contg. B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin)
IT Haemophilus influenzae
(type b; B subunits of cholera toxin, heat-labile enterotoxin, or verotoxin in vaccines against)
IT **37758-47-7**, Ganglioside GM1 **71965-57-6**, Globotriosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(immunomodulators with signaling activity mediated via binding to)
IT **37758-47-7**, Ganglioside GM1 **71965-57-6**, Globotriosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(immunomodulators with signaling activity mediated via binding to)
RN 37758-47-7 HCAPLUS
CN Ganglioside GM1 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71965-57-6 HCAPLUS
CN Ceramide, 1-O-(O-.alpha.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl)- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 6 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:645848 HCAPLUS
DN 132:132107
TI Lansoprazole decreases peripheral blood monocytes and intercellular adhesion molecule-1-positive mononuclear cells
AU Ohara, Tadashi; Arakawa, Tetsuo
CS Department of Gastroenterology, Sendai Shakai Hoken Hospital, Sendai, 981, Japan
SO Digestive Diseases and Sciences (**1999**), 44(8), 1710-1715
CODEN: DDSCDJ; ISSN: 0163-2116
PB Kluwer Academic/Plenum Publishers
DT Journal
LA English
CC 1-9 (**Pharmacology**)
AB We examd. the effects of lansoprazole, a proton-pump inhibitor, on peripheral blood mononuclear cells in healthy subjects in comparison with ranitidine. Ten healthy volunteers were randomly divided into two groups and given either lansoprazole (30 mg daily for 2 days) or ranitidine (150 mg daily for 21 days). Peripheral blood was collected before and 7, 14,

and 21 days after the start of treatment. Mononuclear cells were isolated by densitometric centrifugation and were examd. for adhesion mols. (ICAM-1, VLA4, **SLex**), membrane markers of the monocyte/macrophage series, and lymphocyte phenotypes. The no. of cells expressing adhesion mols., the no. of monocytes/macrophages, and lymphocyte phenotypes were the same in **Helicobacter pylori**-pos. and -neg. subjects. The no. of cells expressing ICAM-1 was significantly decreased seven days after the start of lansoprazole treatment, and this change persisted until day 14, while ranitidine had no effect. The no. of monocytes (identified by Leu-M3 positivity) was decreased seven days after the start of treatment in both groups, but predominantly in the lansoprazole group. No other changes were obsd. on administration of either drug. These results suggest that short-term treatment with lansoprazole causes persistent inhibition of inflammatory responses irresp. of the presence of **H. pylori** infection. This effect may indicate a possible new mechanism of action of proton-pump inhibitors other than inhibition of acid secretion.

ST proton pump inhibitor lansoprazole intestine inflammation; Helicobacter lansoprazole intestine inflammatory response

IT Cell adhesion molecules
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(ICAM-1 (intercellular adhesion mol. 1); lansoprazole decreases peripheral blood monocytes and intercellular adhesion mol.-1-pos. mononuclear cells and decrease Helicobacter inflammatory responses)

IT Anti-inflammatory agents
Helicobacter pylori
Macrophage
Monocyte
(lansoprazole decreases peripheral blood monocytes and intercellular adhesion mol.-1-pos. mononuclear cells and decrease Helicobacter inflammatory responses)

IT Gastric acid
(secretion; lansoprazole decreases peripheral blood monocytes and intercellular adhesion mol.-1-pos. mononuclear cells and decrease Helicobacter inflammatory responses)

IT 9000-83-3, ATPase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hydrogen ion-translocating inhibitor; lansoprazole decreases peripheral blood monocytes and intercellular adhesion mol.-1-pos. mononuclear cells and decrease Helicobacter inflammatory responses)

IT 103577-45-3, Lansoprazole
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(lansoprazole decreases peripheral blood monocytes and intercellular adhesion mol.-1-pos. mononuclear cells and decrease Helicobacter inflammatory responses)

RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Baak, L; Gut 1991, V32, P977 MEDLINE
(2) Bianchi-Porro, G; Aliment Pharmacol Ther 1994, V8, P541 MEDLINE
(3) Catalano, F; Ital J Gastroenterol 1995, V27, P21 MEDLINE
(4) Cho, C; Gastroenterology 1996, V110, PA81
(5) Crabtree, J; Gut 1991, V32, P1473 MEDLINE
(6) Crabtree, J; J Clin Pathol 1994, V47, P61 MEDLINE
(7) Crabtree, J; Scand J Immunol 1993, V37, P65 HCAPLUS
(8) Fan, X; J Clin Pathol 1995, V48, P133 MEDLINE
(9) Hoshino, E; J Clin Gastroenterol 1995, V20(suppl 2), PS72
(10) Interdisciplinary Group for Ulcer Study; Digestion 1995, V56, P181
(11) Labenz, J; Gastroenterology 1997, V112, P1442 MEDLINE
(12) Moss, S; Gut 1994, V35, P1567 MEDLINE

(13) Noach, L; Scand J Gastroenterol 1994, V29, P425 HCAPLUS
(14) Santucci, L; Gastroenterology 1995, V108, P393 HCAPLUS
(15) Santucci, L; Gastroenterology 1995, V108, PA209
(16) Satoh, M; J Immunol 1996, V157, P3886 HCAPLUS
(17) Savarino, V; Dig Dis Sci 1994, V39, P1473 MEDLINE
(18) Shintani, F; Mol Neurobiol 1995, V10, P47 HCAPLUS
(19) Sugawara, S; J Immunol Methods 1987, V100, P83 HCAPLUS
(20) Suzuki, M; Free Radic Biol Med 1996, V21, P727 HCAPLUS
(21) Suzuki, M; J Clin Gastroenterol 1997, V20(suppl 2), PS93
(22) Watanabe, T; Am J Pathol 1997, V150, P971 HCAPLUS

L125 ANSWER 7 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1999:223015 HCAPLUS
DN 130:249112
TI Methods and compositions for binding hematopoietic stem cells using a binding partner for sialylated lactosamines on stem cell surfaces
IN Magnani, John L.
PA Glycotech Corporation, USA
SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C12N005-06
ICS C12N005-08; A61K047-48; A61K049-00; B01D015-08; C12Q001-04; G01N033-53
CC 9-2 (Biochemical Methods)
Section cross-reference(s): 13, **63**
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9915628	A1	19990401	WO 1998-US20063	19980924 <--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2304961	AA	19990401	CA 1998-2304961	19980924 <--
	AU 9895089	A1	19990412	AU 1998-95089	19980924 <--
	EP 1017790	A1	20000712	EP 1998-948540	19980924 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001517427	T2	20011009	JP 2000-512922	19980924 <--
PRAI	US 1997-59972P	P	19970925 <--		
	WO 1998-US20063	W	19980924 <--		

AB Methods and compns. are provided for binding hematopoietic stem cells. The methods generally employ a binding partner that forms a complex with a sialylated lactosamine structure present on the surface of stem cells. The formation of such complexes facilitates, for example, immobilization, purifn., identification and targeting of hematopoietic stem cells. The compns. described herein generally comprise a binding partner, which may be free, attached to a support material or linked to a label or therapeutic agent. Hematopoietic stem cells were immobilized in microtiter plate wells contg. monoclonal antibody NUH2, Maackia amurensis lectin, tomato lectin, and sialoadhesin, but not to bovine serum albumin or IgM.
ST binding hematopoietic stem cell sialylated lactosamine; immobilization hematopoietic stem cell antibody lectin sialoadhesin; drug targeting hematopoietic stem cell
IT Cytometry
(FACS (fluorescence-activated cell sorting); methods and compns. for

binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT CD34 (antigen)
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(antibodies to, in FACS anal.; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Agglutinins and Lectins
Antibodies
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Drugs
Toxicants
(binding partner assocd. with; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Polynucleotides
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(binding partner assocd. with; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Bags
(binding partner attached to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT **Helicobacter pylori**
(carbohydrate receptor of, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(carbohydrate, of **Helicobacter pylori**, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Reagents
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(detection, kits contg.; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Animal tissue culture
(dish for, binding partner attached to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

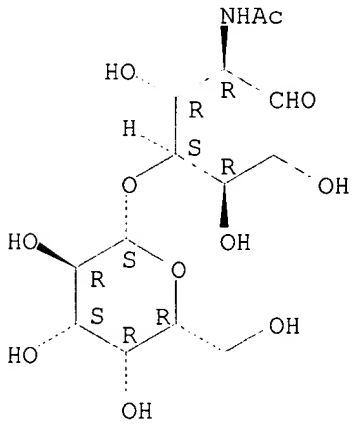
IT Agglutinins and Lectins
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(galactose-binding, galectins, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)

IT Blood
Bone marrow
Cord blood
(hematopoietic stem cells of; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated

lactosamines on stem cell surfaces)
IT Bioreactors
Bioreactors
(hollow-fiber membrane, binding partner attached to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Immunoassay
(immunofluorescent staining; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Immobilization, biochemical
(kit for; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Bacteria (Eubacteria)
Elder (Sambucus nigra)
Erythrina crista-galli
Maackia amurensis
Mammal (Mammalia)
Plant (Embryophyta)
Tomato
Wheat
(lectin of, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Drug delivery systems
Test kits
(methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Antibodies
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(monoclonal, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Carbohydrates, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(receptors, of **Helicobacter pylori**, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Adhesins
RL: BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(sialoadhesins, as binding partner; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Hematopoietic precursor cell
(stem; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT Chromatography
(supports, binding partner attached to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT 83563-61-5
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(binding partner to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
IT **32181-59-2D**, sialyl-terminated

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(repeating unit, binding partner to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Anon; US 5227160 A HCAPLUS
(2) Baxter International Inc; WO 9425571 A 1994 HCAPLUS
(3) The Biomembrane Institute; EP 0351045 A 1990 HCAPLUS
IT **32181-59-2D**, sialyl-terminated
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(repeating unit, binding partner to; methods and compns. for binding hematopoietic stem cells using binding partner for sialylated lactosamines on stem cell surfaces)
RN 32181-59-2 HCAPLUS
CN D-Glucose, 2-(acetylamino)-2-deoxy-4-O-.beta.-D-galactopyranosyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L125 ANSWER 8 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:653534 HCAPLUS
DN 129:271521
TI Encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy
IN Morrow, Casey D.; Porter, Donna C.; Ansardi, David C.
PA The UAB Research Foundation, USA
SO U.S., 62 pp., Cont.-in-part of U.S. Ser. No. 87,009, abandoned.
CODEN: USXXAM
DT Patent
LA English
IC ICM C12N015-43
ICS C12P021-02; A61K039-13
NCL 435320100
CC 3-2 (Biochemical Genetics)
Section cross-reference(s): **1**, **63**
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5817512	A	19981006	US 1995-389459	19950215 <--
	US 5622705	A	19970422	US 1995-444882	19950519 <--
	US 5614413	A	19970325	US 1996-589446	19960122 <--
	WO 9625173	A1	19960822	WO 1996-US1895	19960213 <--

W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN

AU 9649784 A1 19960904 AU 1996-49784 19960213 <--
EP 809513 A1 19971203 EP 1996-906392 19960213 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE
US 6063384 A 20000516 US 1997-987867 19971209 <--
US 2002051768 A1 20020502 US 2001-756551 20010108 <--
PRAI US 1993-87009 B2 19930701 <--
US 1995-389459 A 19950215 <--
WO 1996-US1895 W 19960213 <--
US 1997-987867 A1 19971209 <--
US 1999-376184 B1 19990817 <--

AB The present invention pertains to a method of encapsidating a recombinant poliovirus nucleic acid to obtain a yield of encapsidated viruses which substantially comprises encapsidated recombinant poliovirus nucleic acid. The method of encapsidating a recombinant poliovirus nucleic acid includes contacting a host cell with a recombinant poliovirus nucleic acid which lacks the nucleotide sequence encoding at least a portion of a protein necessary for encapsidation and an expression vector comprising a nucleic acid which encodes at least a portion of one protein necessary for encapsidation under conditions appropriate for introduction of the recombinant poliovirus nucleic acid and the expression vector into the host cell and obtaining a yield of encapsidated viruses which substantially comprises an encapsidated recombinant poliovirus nucleic acid. A foreign nucleotide sequence is generally substituted for the nucleotide sequence of the poliovirus nucleic acid encoding at least a portion of a protein necessary for encapsidation. The invention further pertains to encapsidated recombinant poliovirus nucleic acids produced by the method of this invention and compns. contg. the encapsidated or nonencapsidated recombinant poliovirus nucleic acid contg. a foreign nucleotide sequence for use in a method of stimulating an immune response in a subject to the protein encoded by the foreign nucleotide sequence. Encapsidation of recombinant poliovirus nucleic acid contg. the HIV-1 gag or pol gene(s) and use of the recombinant poliovirus to induce immunity against HIV-1 were demonstrated. Vectors expressing carcinoembryonic antigen are also described.

ST gene therapy vaccine poliovirus vector encapsidation; HIV vaccine poliovirus vector encapsidation

IT Vaccines
(AIDS; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)

IT Antigens
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**Helicobacter pylori**; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)

IT Antigens
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(Jen CRG from colorectal and lung cancer cells; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)

IT Antigens
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(Mycobacterium tuberculosis B; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)

IT Polyproteins
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(P1 capsid; encapsidated recombinant viral nucleic acid and vectors for

vaccine and gene therapy)
IT Proteins, specific or class
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**;
BIOL (Biological study); USES (Uses)
(VP1, required for encapsidation; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Proteins, specific or class
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**;
BIOL (Biological study); USES (Uses)
(VP2, required for encapsidation; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Proteins, specific or class
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**;
BIOL (Biological study); USES (Uses)
(VP3; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Proteins, specific or class
RL: BUU (Biological use, unclassified); **THU (Therapeutic use)**;
BIOL (Biological study); USES (Uses)
(VP4; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT **Helicobacter pylori**
Human immunodeficiency virus 1
Influenza virus
Mycobacterium tuberculosis
Respiratory syncytial virus
Rotavirus
(antigen from; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Toxins
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(cholera; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Toxins
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(diphtheria; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene therapy
Plasmid vectors
Virus vectors
(encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Viral RNA
RL: BPN (Biosynthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Antisense DNA
Carcinoembryonic antigen
Cytokines
Envelope proteins
Platelet-derived growth factors
Ribozymes
gag proteins
neu (receptor)
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, microbial
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(env; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, microbial

RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gag; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Enzymes, biological studies
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gene pol; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, microbial
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(neu; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, animal
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oncogene, erb; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, microbial
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(pol; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Gene, microbial
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sis; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Vaccines
(synthetic; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Toxins
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(tetanus; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Anti-AIDS agents
(vaccines; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT Human poliovirus
Vaccinia virus
(vectors; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT 103406-62-8, 2A Proteinase
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(antigen from; encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
IT **19600-01-2**, Ganglioside gm2 **62010-37-1**, Ganglioside gd3
65988-71-8, Ganglioside gd2
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE
(1) Anon; Biotechnology 1994, V12, P128
(2) Ansardi, D; Cancer Research 1994, V54, P6359 HCAPLUS
(3) Ansardi, D; J Cell Biochem Suppl 1993, V17(D), P22
(4) Ansardi, D; J Virol 1991, V65(4), P2088 HCAPLUS
(5) Ansardi, D; J Virol 1992, V66(7), P4556 HCAPLUS
(6) Ansardi, D; J Virol 1993, V67(6), P3684 HCAPLUS
(7) Choi; J Virol 1991, V65, P2875 HCAPLUS
(8) Choi, W; J Virol 1991, V65(6), P2875 HCAPLUS
(9) Evans, D; Nature 1989, V339, P385 HCAPLUS
(10) Haynes, B; Science 1993, V260, P1279 MEDLINE
(11) Jenkins, O; J Virol 1990, V64(3), P1201 HCAPLUS
(12) Kantor; J Natl Cancer Institute 1992, V84, P1084 MEDLINE
(13) Knuth; Current Opinion in Immunol 1991, V3, P659 HCAPLUS
(14) McGhee, J; AIDS Research Reviews, Ch 15 1992, V2, P289

(15) Moldoveanu, Z; FASEB J 1995, V9(3, 1247), PA214
(16) Morrow, C; AIDS Research and Human Retroviruses 1994, V10(Suppl 1), PS61
(17) Percy, N; J Virol 1992, V66(8), P5040 HCAPLUS
(18) Porter, D; J Cell Biochem Suppl 1993, V17(D), P26
(19) Porter, D; J Virol 1993, V67(7), P3712 HCAPLUS
(20) Porter, D; Journal of Virology 1995, V69(3), P1548 HCAPLUS
(21) Porter, D; Virus Research 1993, V29, P241 HCAPLUS
IT **19600-01-2**, Ganglioside gm2 **62010-37-1**, Ganglioside gd3
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(encapsidated recombinant viral nucleic acid and vectors for vaccine and gene therapy)
RN 19600-01-2 HCAPLUS
CN Ganglioside GM2 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 62010-37-1 HCAPLUS
CN Ganglioside GD3 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 9 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:619952 HCAPLUS
DN 130:38607
TI Epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus
AU Miller-Podraza, Halina; Larsson, Thomas; Nilsson, Jonas; Teneberg, Susann; Matrosovich, Mikhail; Johansson, Lena
CS Department of Medical Biochemistry, Goteborg University, Goteborg, S-413 90, Swed.
SO Acta Biochimica Polonica (**1998**), 45(2), 439-449
CODEN: ABPLAF; ISSN: 0001-527X
PB Polish Biochemical Society
DT Journal
LA English
CC 33-8 (Carbohydrates)
Section cross-reference(s): 15
AB Receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus were chem. modified and analyzed by neg. ion fast atom bombardment mass spectrometry (FAB MS) or electron ionization mass spectrometry (EI MS) after per-methylation. Derivatizations included mild periodate oxidn. of the sialic acid glycerol tail or conversion of the carboxyl group to primary alc. or amides. The modified gangliosides were then tested for binding affinity using thin-layer plates overlaid with labeled microbes or microbe-derived proteins. Mild periodate oxidn., which shortens sialic acid tail without destruction of sugar cores, abolished or drastically reduced binding of **H. pylori** and avian influenza virus to sialyl-3-para-globoside (S-3-PG). The same effect was obsd. in the case of binding of the human influenza virus to receptor-active gangliosides of human leukocytes. Conversion of S-3-PG or leukocyte gangliosides to primary alcs. or amides also abolished the binding. However, mild periodate oxidn. had no effect on binding of NAP (neutrophil-activating protein of **H. pylori**) to the active ganglioside.
ST ganglioside receptor activity modification **Helicobacter pylori** influenza virus; amide primary alc prepn ganglioside oxidn redn
IT Peroxidation
(biol.; epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)
IT Carboxyl group
Epitopes
Helicobacter pylori

Influenza virus
Leukocyte
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT Receptors
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT Gangliosides
Sialic acids
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT Amides, preparation
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT Alcohols, preparation
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); RCT (Reactant); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent)
(primary; epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT **71833-58-4**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT **71833-57-3**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

IT **71833-57-3DP**, oxidized and reduced **216768-01-3P 216768-02-4P 216768-03-5P 216768-04-6P 216768-05-7P 216768-06-8P**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD

RE

(1) Bergelson, L; Eur J Biochem 1982, V128, P467 HCAPLUS
(2) Curatolo, W; Biochim Biophys Acta 1987, V906, P37
(3) Evans, D; Infect Immun 1988, V56, P2896 HCAPLUS
(4) Evans, D; Infect Immun 1995, V63, P2213 HCAPLUS
(5) Evans, D; Methods Enzymol 1995, V253, P336 HCAPLUS
(6) Fishman, P; Chem Phys Lipids 1986, V42, P137 HCAPLUS
(7) Goldstone, A; J Pathol 1996, V179, P129 MEDLINE
(8) Handa, S; J Biochem (Tokyo) 1984, V95, P1323 HCAPLUS
(9) Herrler, G; Biology of the Sialic Acids 1995, P315 HCAPLUS
(10) Hirmo, S; Glycoconjugate J 1996, V13, P1005 HCAPLUS
(11) Karlsson, K; Annu Rev Biochem 1989, V58, P309 HCAPLUS

(12) Karlsson, K; Methods Enzymol 1987, V138, P212 HCAPLUS
(13) Kelm, S; Eur J Biochem 1992, V205, P147 HCAPLUS
(14) Koscielak, J; Eur J Biochem 1976, V71, P9 HCAPLUS
(15) Koscielak, J; Eur J Biochem 1979, V96, P331 HCAPLUS
(16) Lanne, B; Biochemistry 1995, V34, P1845 HCAPLUS
(17) Lelwala-Guruge, J; APMIS 1992, V100, P908 MEDLINE
(18) Liukkonen, J; J Biol Chem 1992, V267, P21105 HCAPLUS
(19) Loomes, L; Nature 1984, V307, P560 HCAPLUS
(20) Matrosovich, M; Virology 1993, V196, P111 HCAPLUS
(21) Matrosovich, M; Virology 1996, V223, P413 HCAPLUS
(22) Matrosovich, M; Virology 1997, V233, P224 HCAPLUS
(23) Miller-Podraza, H; Biochim Biophys Acta 1993, V1168, P330 HCAPLUS
(24) Miller-Podraza, H; Glycoconjugate J 1996, V13, P453 HCAPLUS
(25) Miller-Podraza, H; Glycoconjugate J 1997, V14, P231 HCAPLUS
(26) Miller-Podraza, H; Glycoconjugate J 1997, V14, P467 HCAPLUS
(27) Miller-Podraza, H; Infect Immun 1997, V65, P2480 HCAPLUS
(28) Muthing, J; Carbohydrate Res 1996, V290, P217 MEDLINE
(29) Nakamura, K; J Biochem 1986, V99, P219 HCAPLUS
(30) Nedrud, J; Curr Opin Gastroenterol 1996, V12, P62
(31) Rogers, G; Virology 1989, V173, P317 HCAPLUS
(32) Simon, P; Infect Immun 1997, V65, P750 HCAPLUS
(33) Slomiany, B; Biochem Int 1989, V19, P929 HCAPLUS
(34) Suzuki, Y; J Biol Chem 1985, V260, P1362 HCAPLUS
(35) Suzuki, Y; Virology 1992, V189, P121 HCAPLUS
(36) Svennerholm, L; INSERM 1984, V126, P21
(37) Teneberg, S; J Biol Chem 1997, V272, P19067 HCAPLUS
(38) Toshihito, S; FEBS Lett 1991, V282, P385
(39) Varki, A; Glycobiology 1992, V2, P25 HCAPLUS
(40) Veh, R; Biochim Biophys Acta 1977, V486, P145 HCAPLUS
(41) Wadstrom, T; Curr Opin Gastroenterol 1995, V11, P69
(42) Zdebska, E; Carbohydrate Res 1983, V120, P113 HCAPLUS

IT **71833-58-4**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

RN 71833-58-4 HCAPLUS

CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **71833-57-3**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); RACT (Reactant or reagent)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)

RN 71833-57-3 HCAPLUS

CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

IT **71833-57-3DP**, oxidized and reduced **216768-01-3P 216768-02-4P 216768-03-5P 216768-04-6P 216768-05-7P 216768-06-8P**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL

(Biological study); PREP (Preparation)
(epitope dissection of receptor-active gangliosides with affinity for **Helicobacter pylori** and influenza virus)
RN 71833-57-3 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-01-3 HCAPLUS
CN Ceramide, 1-O-[O-5-(acetylamino)-3,5-dideoxy-D-glycero-.alpha.-D-galacto-2-nonulopyranosyl-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-02-4 HCAPLUS
CN Ceramide, 1-O-[O-(N5-acetyl-N1-ethyl-.alpha.-neuraminamidosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-03-5 HCAPLUS
CN Ceramide, 1-O-[O-(N5-acetyl-N1-methyl-.alpha.-neuraminamidosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-04-6 HCAPLUS
CN Ceramide, 1-O-[O-(N5-acetyl-.alpha.-neuraminamidosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-05-7 HCAPLUS
CN Ceramide, 1-O-[O-[N5-acetyl-N1-(phenylmethyl)-.alpha.-neuraminamidosyl]-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 216768-06-8 HCAPLUS
CN Ceramide, 1-O-[O-(N5-acetyl-N1-propyl-.alpha.-neuraminamidosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 10 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:608540 HCAPLUS
DN 129:225715
TI Antibiotic-ligand conjugates and methods of use thereof
IN Lingwood, Clifford A.
PA HSC Research and Development Limited Partnership, Can.
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent

LA English
IC ICM A61K047-48
CC 1-5 (**Pharmacology**)
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9837915	A1	19980903	WO 1998-CA142	19980226	<--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
	AU 9860845	A1	19980918	AU 1998-60845	19980226	<--
	WO 9943356	A1	19990902	WO 1998-CA817	19980826	<--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ					
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
	AU 9889679	A1	19990915	AU 1998-89679	19980826	<--
PRAI	US 1997-39160P	P	19970226	<--		
	US 1998-30095	A	19980225	<--		
	WO 1998-CA142	W	19980226	<--		
	US 1998-95673P	P	19980807	<--		
	WO 1998-CA817	W	19980826	<--		

AB Methods for treating a glycolipid-mediated state in a subject are described. An effective amt. of .gtoreq.1 therapeutic compd. A-B, in which A is a glycolipid receptor moiety and B is an active agent, is administered to a subject, such that treatment of the glycolipid mediated state occurs. Methods also include administering and effective amt. of .gtoreq.1 therapeutic compd., or a pharmaceutically acceptable salt thereof, to a subject such that a disease state assocd. with a shiga-like toxin (SLT) is treated. Packaged pharmaceutical compns. for treating SLTs are described. The package includes a container for holding an effective amt. of a pharmaceutical compn. and instructions for using the pharmaceutical compn. for treatment of SLT. The pharmaceutical compn. includes at least one therapeutic compd. for modulating a SLT in a subject.

ST antibiotic ligand conjugate glycolipid mediated condition; receptor glycolipid active agent conjugate therapeutic; shiga like toxin antibiotic ligand conjugate

IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Shiga-like toxin I; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Shiga-like toxin II; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Shiga-like toxin, III; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Toxins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Shiga-like toxin; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Antibacterial agents
Antimicrobial agents
Borrelia burgdorferi
Burkholderia cepacia
Chlamydia pneumoniae
Chlamydia trachomatis
Clostridium difficile
Clostridium perfringens
Coxiella burnetii
Drug delivery systems
Escherichia coli
Haemophilus influenzae
Haemophilus parainfluenzae
Helicobacter pylori
Klebsiella pneumoniae
Moraxella catarrhalis
Mycobacterium intracellulare
Mycobacterium tuberculosis
Neisseria gonorrhoeae
Neisseria meningitidis
Pasteurella multocida
Pathogen
Pseudomonas aeruginosa
Salmonella typhimurium
Shigella dysenteriae
Shigella flexneri
Staphylococcus aureus
Stenotrophomonas maltophilia
Streptococcus agalactiae
Streptococcus pneumoniae
(antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Glycolipids
Phosphatidylethanolamines, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Oligosaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(ceramide conjugates, conjugates with active agents; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(cytotoxins; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Antibiotics
Drugs
(glycolipid receptor conjugates; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Cyclic compounds
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(glycolipid receptor conjugates; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Receptors
RL: **BAC (Biological activity or effector, except adverse)**; BPR (Biological process); BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PROC (Process); USES (Uses)
(glycolipid, active agent conjugates; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Envelope proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(gp120env; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT Ceramides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharide conjugates, conjugates with active agents; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT 260-94-6D, Acridine, derivs., glycolipid receptor conjugates 281-23-2D, Adamantane, derivs., glycolipid receptor conjugates 1406-05-9D, Penicillin, glycolipid receptor conjugates **35960-33-9D**, active agent conjugates 66580-68-5, Globotriaose 66580-68-5D, Globotriaose, adamantyl and acridine derivs. **71012-19-6D**, N-acyl derivs., active agent conjugates 212699-22-4 212699-23-5
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT 11111-12-9D, Cephalosporin, glycolipid receptor conjugates
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(cephalosporin antibiotics; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT 25795-42-0D, Cepham, glycolipid receptor conjugates
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(cepham antibiotics; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT 1546-79-8 24909-72-6, Oleic anhydride 103213-60-1, Erucic anhydride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction; antibiotic-ligand conjugates for treatment of glycolipid-mediated states)

IT 56739-51-6
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction; kntibiotic-ligand conjugates for treatment of glycolipid-mediated states)

RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE

(1) Arab, S; ONCOL RES 1997, V9(10), PP553
(2) Bitzan, M; J INFECT DIS 1998, V177(4), PP955
(3) Hostetler, K; US 5463092 A 1995 HCAPLUS
(4) Krivan, H; US 5466681 A 1995 HCAPLUS
(5) Krivan, H; US 5696000 A 1997 HCAPLUS
(6) Krivan, H; WO 9202817 A 1997 HCAPLUS
(7) Leffler, H; US 4464360 A 1984 HCAPLUS
(8) Lingwood, C; GLYCOCONJUGATE JOURNAL 1996, V13(4), P495 HCAPLUS
(9) Liposome Co Inc; WO 8911272 A 1989 HCAPLUS
(10) Microcarb Inc; WO 9211015 A 1992 HCAPLUS
(11) Univ Montana Res Dev Inst; WO 9718790 A 1997 HCAPLUS

IT **35960-33-9D**, active agent conjugates **71012-19-6D**, N-acyl derivs., active agent conjugates
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(antibiotic-ligand conjugates for treatment of glycolipid-mediated states)
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 11 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:469511 HCAPLUS
DN 129:243840
TI Serum antibody response against **Helicobacter pylori** NCTC 11637 smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy
AU Pece, S.; Messa, C.; Caccavo, D.; Giuliani, G.; Greco, B.; Fumarola, D.; Berloco, P.; Di Leo, A.; Jirillo, E.; Moran, A. P.
CS Department of Internal Medicine, Immunology and Infectious Diseases, University of Bari, Bari, I-70124, Italy
SO Journal of Endotoxin Research (**1997**), 4(6), 383-390
CODEN: JENREB; ISSN: 0968-0519
PB Churchill Livingstone
DT Journal
LA English
CC 15-3 (Immunochemistry)
Section cross-reference(s): 14
AB The antigenicity of the **H. pylori** lipopolysaccharide (LPS) mol. during the course of natural **H. pylori** infection in humans was investigated. The IgG and IgA responses against smooth (S)- and rough (R)-form LPS were evaluated in **H. pylori** pos. patients with chronic gastritis (CG) and duodenal ulcer disease (DU), and in **H. pylori**-neg. dyspeptic subjects. The results demonstrated that anti **H. pylori** LPS IgG and IgA antibody levels were enhanced in both groups of **H. pylori**-pos. patients compared with **H. pylori** -neg. subjects, thus confirming that **H. pylori** LPS is part of the immunogenic antigen profile of the bacterium. In addn., a marked response against R-LPS, which correlated with that obsd. against S-LPS, was found for both IgG and IgA, thus indicating that core oligosaccharide plays a powerful immunogenic role. Since the O-side chain of LPS from **H. pylori** NCTC 11637 contains epitopes which mimic Lewis x (Lex) antigens, the presence of antibodies to monomeric, trimeric, and polymeric Lex was also investigated. Antibodies against polymeric Lex were detected in 2 patients suffering from chronic atrophic gastritis and active chronic gastritis, resp.
ST antibody Helicobacter lipopolysaccharide rough smooth gastropathy
IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(A; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)

IT Immunoglobulins
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(G; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT **Blood-group substances**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(**Lex**; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT **Helicobacter pylori**
(antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT Infection
(bacterial; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT Lipopolysaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(bacterial; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT Stomach, disease
(chronic gastritis; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)
IT Intestine, disease
(duodenum, ulcer; antibody response against **Helicobacter pylori** smooth- and rough-lipopolysaccharide phenotypes in patients with **H. pylori**-related gastropathy)

L125 ANSWER 12 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:222395 HCAPLUS
DN 128:321033
TI Inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by bovine colostrum
AU Bitzan, Martin M.; Gold, Benjamin D.; Philpott, Dana J.; Huesca, Mario; Sherman, Philip M.; Karch, Helge; Lissner, Reinhard; Lingwood, Clifford A.; Karmali, Mohamed A.
CS Division of Microbiology, University of Toronto, Ontario, Can.
SO Journal of Infectious Diseases (**1998**), 177(4), 955-961
CODEN: JIDIAQ; ISSN: 0022-1899
PB University of Chicago Press
DT Journal
LA English
CC 18-7 (Animal Nutrition)
Section cross-reference(s): **1**, 15
AB **Helicobacter pylori**, the etiol. agent of chronic-active gastritis and duodenal ulcers in humans, and Helicobacter mustelae, a gastric pathogen in ferrets, bind to phosphatidylethanolamine (PE), a constituent of host gastric mucosal cells, and to gangliotetraosylceramide (Gg4) and gangliotriaosylceramide (Gg3). The effect of a bovine colostrum conc. (BCC) on the interaction of **H. pylori** and H. mustelae to their lipid receptors was examd. BCC blocked attachment of both species to Gg4, Gg3, and PE. Partial inhibition of binding was obsd. with native bovine and human colostra. BCC lacked detectable antibodies (by immunoblotting) to **H. pylori** surface proteins (adhesins). However, colostral lipid exts. contained PE and lyso-PE that bound **H. pylori** in

vitro. These results indicate that colostrum can block the binding of Helicobacter species to select lipids and that binding inhibition is conferred, in part, by colostral PE or PE derivs. Colostral lipids may modulate the interaction of **H. pylori** and other adhesin-expressing pathogens with their target tissues.

ST colostrum lipid receptor helicobacter antimicrobial

IT Stomach, disease
(gastritis; inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT Antimicrobial agents
Colostrum
Helicobacter mustelae
Helicobacter pylori
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT Antibodies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT Adhesins
Phosphatidylethanolamines, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lipid; inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT **35960-33-9**, Gangliotriaosylceramide **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT **35960-33-9**, Gangliotriaosylceramide **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

RN 35960-33-9 HCAPLUS

CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 71012-19-6 HCAPLUS

CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 13 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 1998:115869 HCAPLUS

DN 128:226251

TI Gangliosides as inhibitors for **Helicobacter pylori**

adhesion and interleukin-8 formation
IN Murakami, Motoyasu; Hata, Yoshiyuki
PA Murakami, Motoyasu, Japan; Kaken Pharmaceutical Co., Ltd.
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
IC ICM A61K031-70
ICS A61K031-70
CC 1-9 (**Pharmacology**)
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 10045602	A2	19980217	JP 1996-202098	19960731 <--
PRAI JP 1996-202098		19960731 <--		

AB Gangliosides (GD3, GD1a, GD1b, etc.) are claimed as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation for treatment of stomach diseases including gastritis and ulcer.
ST ganglioside Helicobacter adhesion IL8 antiulcer gastritis
IT Adhesion, biological
Antiulcer agents
Helicobacter pylori
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT Gangliosides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT Interleukin 8
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT Stomach, disease
(gastritis; gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT **71012-19-6**, Asialo-Ganglioside GM1 **89678-50-2**, Ganglioside GM3 **98743-26-1** **103220-36-6**, Ganglioside GM1 inner ester **104443-57-4**, Ganglioside GM2 **104443-58-5**, Ganglioside GT1b **104443-59-6**, GD1a **104443-60-9**, GD1b **104443-61-0**, GD3 **104443-62-1**, Ganglioside GM1 **105732-59-0**, Ganglioside GQ1b **107371-09-5**, Ganglioside GD2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT **71012-19-6**, Asialo-Ganglioside GM1 **89678-50-2**, Ganglioside GM3 **98743-26-1** **103220-36-6**, Ganglioside GM1 inner ester **104443-57-4**, Ganglioside GM2 **104443-58-5**, Ganglioside GT1b **104443-59-6**, GD1a **104443-60-9**, GD1b **104443-61-0**, GD3 **104443-62-1**, Ganglioside GM1 **105732-59-0**, Ganglioside GQ1b **107371-09-5**, Ganglioside GD2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
RN 71012-19-6 HCAPLUS

CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 89678-50-2 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 98743-26-1 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-9-O-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 103220-36-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]-, intramol. 1B1,2B-ester (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-57-4 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-58-5 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-59-6 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-60-9 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-61-0 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-62-1 HCAPLUS

CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 105732-59-0 HCAPLUS

CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 107371-09-5 HCAPLUS

CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-[2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 14 OF 23 HCAPLUS COPYRIGHT 2003 ACS

AN 1997:745956 HCAPLUS

DN 128:30403

TI Bismuth salts of sialyloligosaccharides and a method for treating and inhibiting gastric and duodenal ulcers using them

IN Swarz, Herbert

PA Neose Technologies, Inc., USA

SO PCT Int. Appl., 41 pp.
CODEN: PIXXD2

DT Patent

LA English

IC ICM A61K031-70
ICS A61K031-715; A61K033-24

CC 1-9 (**Pharmacology**)
Section cross-reference(s): **63**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9741875	A1	19971113	WO 1997-US6376	19970428 <--
	W: AU, CA, JP, KR, MX				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	CA 2253913	AA	19971113	CA 1997-2253913	19970428 <--
	AU 9727326	A1	19971126	AU 1997-27326	19970428 <--
	AU 710576	B2	19990923		
	EP 918526	A1	19990602	EP 1997-921225	19970428 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000509714	T2	20000802	JP 1997-539929	19970428 <--
	KR 2000010732	A	20000225	KR 1998-708842	19981102 <--
PRAI	US 1996-16765P	P	19960503 <--		
	WO 1997-US6376	W	19970428 <--		

AB A method for treating and/or inhibiting gastric and duodenal ulcers comprises administering a pharmaceutical compn. comprising a bismuth salt of an oligosaccharide (NeuAc-.alpha.(2-3)-pGal-.beta.(1)-(X)m-(Y)n-)p-Z, (X = bond or group capable of linking pGal to either linking group Y or multivalent support Z; C1 glycosidic O of galactose may be replaced by N, S, C; Y = linking group; Z = multivalent support; m, n = 0, 1; p = 2-1000) is described. Also described is a method for treating and/or inhibiting gastric and duodenal ulcers, comprising administering a pharmaceutical compn. comprising a bismuth salt of an oligosaccharide

NeuAc-.alpha.(2-3)-pGal-.beta.(1)-A (A = group capable of bonding to pGal; C1 glycosidic O of galactose may be replaced by N, S, C).

ST sialyloligosaccharide bismuth salt ulcer inhibitor; gastric ulcer inhibitor sialyloligosaccharide bismuth salt; duodenal ulcer inhibitor sialyloligosaccharide bismuth salt

IT Antihistamines
(H2; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Leb**, **Leb** active oligosaccharide;
sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT Dendritic polymers
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Avidins
Lipids, biological studies
Polysaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates, with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Antiulcer agents
(duodenal; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Intestine
(duodenum, **H. pylori** infection; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Drug delivery systems
(enteric; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Stomach, disease
Stomach, disease
(infection, **H. pylori**; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Emulsions
(lipid, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Drug delivery systems
(liposomes, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Drug delivery systems
(oral; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Alcohols, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(polyhydric, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Antiulcer agents
Drug delivery systems

Helicobacter pylori
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Fetuins
Sialooligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Antibacterial agents
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT Antibiotics
Oligosaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT 12408-02-5, Hydrogen ion, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proton pump inhibitors; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT 63-42-3, Lactose 7440-69-9D, Bismuth, salts with sialyloligosaccharides, biological studies 9003-05-8D, Polyacrylamide, conjugates with sialyloligosaccharide bismuth salts 9004-54-0D, Dextran, conjugates with sialyloligosaccharide bismuth salts, biological studies 12619-70-4D, Cyclodextrin, conjugates with sialyloligosaccharide bismuth salts 25104-18-1D, Polylysine, conjugates with sialyloligosaccharide bismuth salts **35890-38-1**, 3'-Sialyllactose **35890-38-1D**, 3'-Sialyllactose, albumin conjugates **35890-39-2**, 6'-Sialyllactose 38000-06-5D, Polylysine, conjugates with sialyloligosaccharide bismuth salts 199612-73-2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

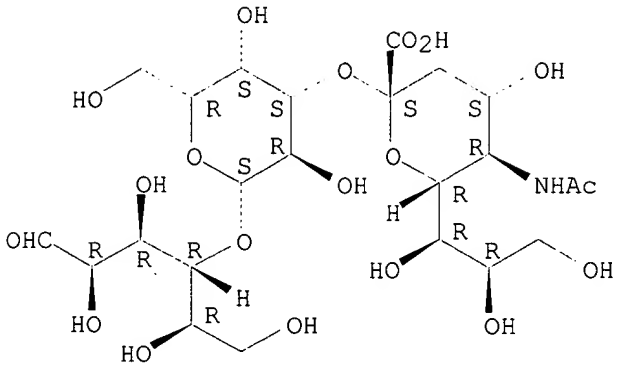
IT 60-54-8D, Tetracycline, derivs. 66357-35-5, Ranitidine 73590-58-6, Omeprazole
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT 12408-02-5, Hydrogen ion, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(transport; inhibitors; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT **35890-38-1**, 3'-Sialyllactose **35890-38-1D**, 3'-Sialyllactose, albumin conjugates **35890-39-2**, 6'-Sialyllactose
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

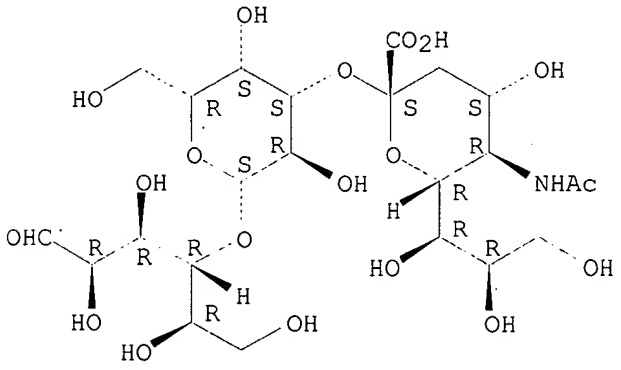
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



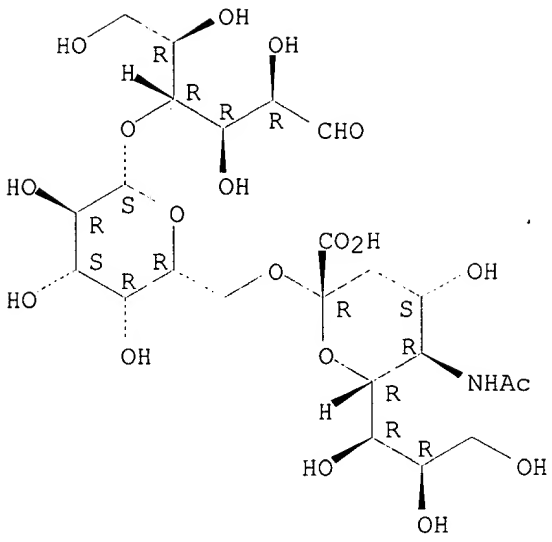
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 35890-39-2 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.6)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L125 ANSWER 15 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:459365 HCAPLUS
DN 127:174735
TI Recognition of glycoconjugates by **Helicobacter pylori**. Comparison of two sialic acid-dependent specificities based on hemagglutination and binding to human erythrocyte glycoconjugates. 2.
AU Miller-Podraza, Halina; Bergstroem, Joergen; Milh, Maan Abul; Karlsson, Karl-Anders
CS Department of Medical Biochemistry, Goteborg University, Goteborg, S-413 90, Swed.
SO Glycoconjugate Journal (**1997**), 14(4), 467-471
CODEN: GLJOEW; ISSN: 0282-0080
PB Chapman & Hall
DT Journal
LA English
CC 14-3 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 10
AB **Helicobacter pylori** expresses sep. binding characteristics depending on growth conditions, as documented by binding to human erythrocyte glycoconjugates. Cells grown in Ham's F12 liq. medium exhibited a selective sialic acid-dependent binding to polyglycosylceramides, PGCs. There was no binding to traditional sialylated glycoconjugates like shorter-chain gangliosides, glycophorin or fetuin. However, cells grown on Brucella agar bound both to PGCs and other sialylated glycoconjugates. Fetuin was an effective inhibitor of hemagglutination caused by agar-grown cells, but had no or a very weak inhibitory effect on hemagglutination by F12-grown bacteria. PGCs were strong inhibitors in both cases, while asialofetuin was completely ineffective. The results indicate that **H. pylori** is able to express two sep. sialic acid-dependent specificities, one represented by binding to fetuin, as described before, and another represented by a selective binding to PGCs.
ST Helicobacter sialoglycoconjugate binding hemagglutination culture condition
IT Culture media
(Brucella agar and Ham's F12 liq. medium; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)
IT Gangliosides

Gangliosides
Glycosphingolipids
Glycosphingolipids
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(asialogangliosides; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT Infection
(bacterial; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT Erythrocyte
Helicobacter pylori
Hemagglutination
(comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT Fetuins
Gangliosides
Glycophorins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT Digestive tract
Digestive tract
(infection; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT Glycoconjugates
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(sialic acid-contg.; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT 9002-18-0, Agar
RL: **BAC (Biological activity or effector, except adverse)**; BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); PROC (Process); USES (Uses)
(Brucella, culture media contg.; comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT 12707-58-3, Ganglioside GD1a 19553-76-5, Ganglioside GD1b **37758-47-7**, Ganglioside GM1 **71833-57-3**, Sialosylparagloboside **110069-38-0**, Ganglioside GT3
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)

IT **37758-47-7**, Ganglioside GM1 **71833-57-3**, Sialosylparagloboside **110069-38-0**, Ganglioside GT3

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(comparison of two sialic acid-dependent specificities of **Helicobacter pylori** based on hemagglutination and binding to human erythrocyte glycoconjugates and dependent on culture growth conditions)
RN 37758-47-7 HCAPLUS
CN Ganglioside GM1 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71833-57-3 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-2-(acetylamino)-2-deoxy-.beta.-D-glucopyranosyl-(1.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 110069-38-0 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 16 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:70383 HCAPLUS
DN 124:114313
TI Role of sulfatides in adhesion of **Helicobacter pylori** to gastric cancer cells
AU Kamisago, Satoshi; Iwamori, Masao; Tai, Tadashi; Mitamura, Keiji; Yazaki, Yoshio; Sugano, Kentaro
CS Third Dep. Internal Medicine, Univ. Tokyo, Tokyo, 113, Japan
SO Infection and Immunity (**1996**), 64(2), 624-8
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 14-7 (Mammalian Pathological Biochemistry)
AB We have demonstrated that clin. isolates of **Helicobacter pylori** preferentially bind to sulfatides (I3SO3-GalCer) and GM3 gangliosides (II3NeuAcLacCer), two predominant acidic glycosphingolipids in the human gastric mucosa, on thin-layer chromatog. plates. However, it has not yet been clarified that these glycosphingolipids truly serve as adhesion receptors for **H. pylori** in live cells. In this study, we used a gastric cancer cell line, KATO III, as a cellular model of **H. pylori** adhesion and examd. the role of sulfatides in attachment. The adhesion of **H. pylori** (i.e., a std. strain of **H. pylori**, NCTC 11637) to KATO III cells and the effects of various substances on this adhesion were monitored and semiquantitated by flow cytometric anal. Sulfated glycoconjugates, such as heparin and gastric mucin, significantly inhibited **H. pylori** adhesion to KATO III cells. Membrane prepns. from KATO III cells strongly inhibited this adhesion. In the membrane prepns., sulfatides were present as a major acidic glycosphingolipid. With the exception of sulfatides, no distinct adhesion of **H. pylori** to glycosphingolipids from KATO III cells was obsd. Moreover, **H. pylori** did not bind to any membrane proteins of KATO III cells. Finally, a monoclonal anti-sulfatide antibody markedly reduced **H. pylori** adhesion to KATO III cells. These results suggest that sulfatides, and possibly related sulfated compds., serve as a major receptor for cell adhesion by **H. pylori**.

ST sulfatide adhesion Helicobacter stomach
IT **Campylobacter pyloridis**
Stomach
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)
IT Sulfatides
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)
IT Mucins
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells inhibition by)
IT Adhesion
(bio-, sulfatides in adhesion of **Helicobacter pylori** to gastric cells)
IT **54827-14-4**, Ganglioside gm3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)
IT 9005-49-6, Heparin, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells inhibition by)
IT **54827-14-4**, Ganglioside gm3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)
RN 54827-14-4 HCAPLUS
CN Ganglioside GM3 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 17 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:13184 HCAPLUS
DN 124:76496
TI Asialoganglioside-antibiotic conjugates for treating bacterial infection
IN Krivan, Howard C.; Blomberg, A. Lennart I.
PA MicroCarb, Inc., USA
SO U.S., 12 pp. Cont. of U.S. Ser. No. 484,568, abandoned.
CODEN: USXXAM
DT Patent
LA English
IC ICM A61K031-715
ICS A61K031-705; A61K039-00
NCL 514054000
CC 1-5 (**Pharmacology**)
Section cross-reference(s): 2, 15, **63**
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5466681	A	19951114	US 1994-180397	19940112 <--
PRAI US 1990-484568		19900223 <--		

AB Asialogangliosides, such as asialo-GM1 and asialo-GM2, are used for targeting penicillin antibiotics to bacteria. The present invention provides prepn. of conjugates of the microorganism receptor (i.e. asialo-GM1 and asialo-GM2) and anti-infectives (i.e. antibiotic, steroid, synthetic drugs, or a mol. that can induce prodn. of antibody). The present invention also provides methods for treating infections in warm-blooded animals due to pathogenic microorganisms, e.g. Streptococcus pneumoniae, **Helicobacter pylori**.

ST asialoganglioside antibiotic conjugate bacterial infection
IT Antibiotics
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with asialoganglioside; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT Bacteria
Campylobacter pyloridis
Microorganism
Streptococcus pneumoniae
(infection; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT Gangliosides
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(asialo-, conjugates with antibiotics; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT 131070-85-4P 131070-86-5P 131070-89-8P 131070-90-1P 131070-91-2P
131070-92-3P 131083-69-7P 147662-10-0P 147662-11-1P 147780-81-2P
172723-15-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT **71012-19-6DP**, Asialo-GM1, conjugates with amoxicillin
172723-16-9P
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT 1406-05-9D, Penicillin, conjugates with asialoganglioside 26787-78-0D, Amoxicillin, conjugates with asialoganglioside **35960-33-9D**, Asialo-GM2, conjugates with antibiotic
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
IT **71012-19-6DP**, Asialo-GM1, conjugates with amoxicillin
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **35960-33-9D**, Asialo-GM2, conjugates with antibiotic
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 18 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:995045 HCAPLUS
DN 124:146728
TI Preparation of synthetic carbohydrate which bind to **Helicobacter pylori** for use as drugs.
IN Danishefsky, Samuel J.; Randolph, John T.
PA Sloan-Kettering Institute for Cancer Research, USA

SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07H005-02
ICS C07H015-02; C07H015-20; A61K031-715; A61K031-72
CC 33-4 (Carbohydrates)
Section cross-reference(s): 1
FAN.CNT 7

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9525113	A1	19950921	WO 1995-US3273	19950315 <--
	W: AU, CA, JP, MX				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 5543505	A	19960806	US 1994-213053	19940315 <--
	AU 9521005	A1	19951003	AU 1995-21005	19950315 <--
PRAI	US 1994-213053	A	19940315 <--		
	WO 1995-US3273	W	19950315 <--		

OS MARPAT 124:146728
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Title compds. (I; A = amino acid bearing an .omega.-amino group or .omega.-carbonyl group, amino acid residue of a peptide which bears an .omega.-amino group or .omega.-carbonyl group, amino acid residue of a protein which bears an .omega.-amino group or .omega.-carbonyl group; R1 = H, OH, NH2, NHR4; R4 = SO2Ph, alkyl, acyl, aryl; M = Q1; n = 0-18; where n is >1, each M is independently the same or different; p = 0, 1; R2, R3, R5, R6 = H, OH; with the proviso that geminal R2 and R3 are not both OH and geminal R5 and R6 are not both OH; X, Y = H2, O; q .gtoreq.1; with the proviso than when A = amino acid bearing an .omega.-amino group or an .omega.-carbonyl group, q = 1), are claimed for treatment of disorders caused by **Helicobacter pylori** (no data). Thus, conjugatable Lewis Y blood group determinant (II) was prepd. in several steps from lactal (III) via intermediate (IV).

ST oligosaccharide prepn **helicobacter pylori** adhesion inhibitor; ulcer inhibitor oligosaccharide prepn; gastric adenocarcinoma treatment oligosaccharide; blood group determinant conjugatable prepn

IT **Blood-group substances**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(conjugatable **Lewis** X and Y determinants; prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT **Campylobacter pyloridis**
Neoplasm inhibitors
Ulcer inhibitors
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT Oligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT Stomach, neoplasm

(adenocarcinoma, treatment; prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 173053-82-2P
RL: PNU (Preparation, unclassified); PREP (Preparation)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 98-10-2, Benzenesulfonamide 65207-55-8 127061-08-9 137915-37-8 142800-26-8 145852-76-2 149625-80-9 149847-26-7D, polymer-bound 159494-42-5 173053-78-6 173053-80-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 159494-36-7P 159494-38-9P 162128-74-7P 162128-75-8P 162128-76-9P 162128-80-5P 162128-81-6P 162128-82-7P 162128-84-9P 162128-85-0P 163228-26-0P 163228-28-2P 163228-34-0P 163228-36-2P 173053-77-5DP, polymer-bound 173053-79-7P 173053-81-1P 173053-84-4DP, polymer-bound 173053-85-5DP, polymer-bound 173053-85-5P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 162128-77-0P 163228-29-3P 173053-83-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

L125 ANSWER 19 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:893094 HCAPLUS
DN 123:276048
TI Oligosaccharides for treating and inhibiting gastric and duodenal ulcers
IN Zopf, David A.; Simon, Paul M.; Roth, Stephen; Mcguire, Edward J.; Langer, Dennis H.
PA Neose Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-715
CC 1-9 (**Pharmacology**)
Section cross-reference(s): **63**

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9523605	A1	19950908	WO 1995-US2388	19950302	<--
	W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TT, UA					
	RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
	CA 2183329	AA	19950908	CA 1995-2183329	19950302	<--
	AU 9519323	A1	19950918	AU 1995-19323	19950302	<--
	AU 709149	B2	19990819			
	EP 749314	A1	19961227	EP 1995-911945	19950302	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE					
	JP 09509931	T2	19971007	JP 1995-522955	19950302	<--
	JP 3179108	B2	20010625			
	US 5514660	A	19960507	US 1995-474199	19950607	<--
	US 5753630	A	19980519	US 1996-598431	19960208	<--
	US 5883079	A	19990316	US 1998-75862	19980512	<--
PRAI	US 1994-204515	A	19940302	<--		
	US 1992-922519	B2	19920731	<--		

US 1993-104483 B1 19930728 <--
WO 1995-US2388 W 19950302 <--
US 1995-474199 A1 19950607 <--
US 1996-598431 A1 19960208 <--

AB A method for treating and/or inhibiting gastric and duodenal ulcers, comprises administering a pharmaceutical compn. comprising an oligosaccharide of the following formula: (NeuAc-.alpha.(2-3)-pGal-.beta.(1)-(-X-)m-(-Y-)n-)p-Z; wherein X is a chem. bond or a group capable of linking the p-galactose to either the linking group Y or the multivalent support Z; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C; Y is a linking group; Z is a multivalent support; m is 0 or 1; n is 0 or 1; and p is an integer of 2-1,000. Also described is a pharmaceutical compn. comprising an oligosaccharide of the formula: NeuAc-.alpha.(2-3)-pGal-.beta.(1)-A wherein A is a group capable of bonding to the p-galactose; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C. IC50 value of 3'-sialyl lactose against **Helicobacter pylori** was 6.times.10-3 mmol/mL.
An antiulcer compn. was prepd. by mixing 1g 3'-sialyl lactose and 0.25g ranitidine in water/propylene glycol.

ST ulcer inhibitor oligosaccharide; antiulcer sialyl lactose Helicobacter inhibitor

IT **Campylobacter pyloridis**
(infections; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Ulcer inhibitors
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Fetuins
Oligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Antibiotics
(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Antihistaminics
(H2, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **Blood-group substances**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**Leb**, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Ulcer inhibitors
(duodenal, oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Pharmaceutical dosage forms
(oral, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Albumins, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(reaction products, with sialyl lactose; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT **35890-38-1**, 3'-Sialyl lactose **35890-38-1D**, 3'-Sialyl lactose, reaction products with albumins **35890-39-2**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)

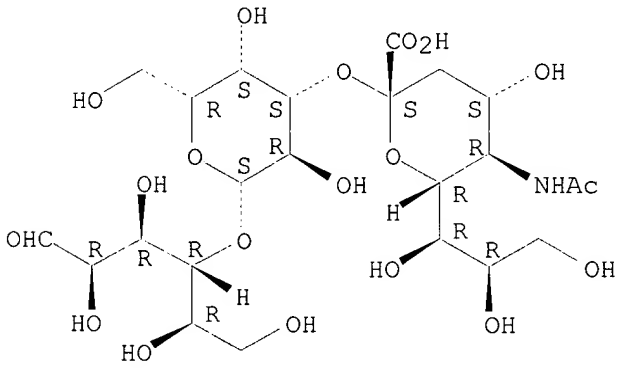
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT 60-54-8, Tetracycline 66357-35-5, Ranitidine 73590-58-6, Omeprazole
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **35890-38-1**, 3'-Sialyl lactose **35890-38-1D**, 3'-Sialyl lactose, reaction products with albumins **35890-39-2**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

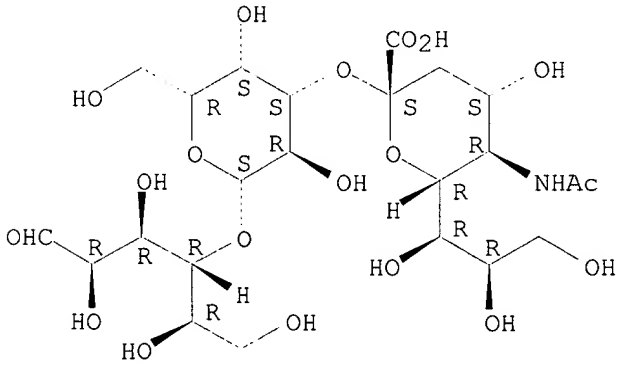
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



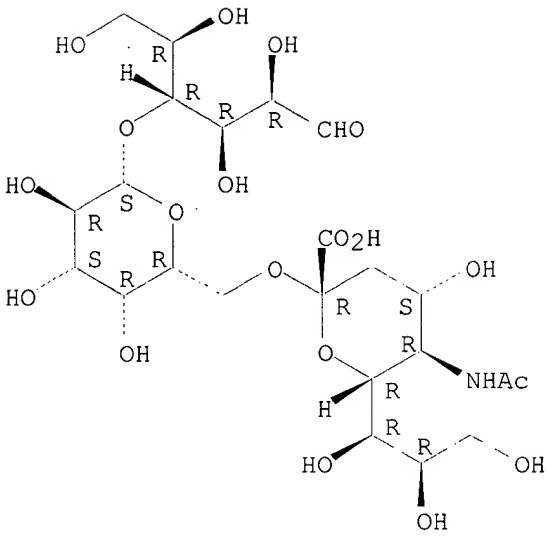
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 35890-39-2 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.6)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L125 ANSWER 20 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:424264 HCAPLUS
DN 122:184826
TI Blood group antigens as tumor markers, parasitic/bacterial/viral receptors, and their association with immunologically important proteins
AU Garratty, G.
CS Research Department, American Red Cross Blood Services, Los Angeles, CA, 90006, USA
SO Immunological Investigations (**1995**), 24(1&2), 213-32
CODEN: IMINEJ; ISSN: 0882-0139
DT Journal; General Review
LA English
CC 15-0 (Immunochemistry)
Section cross-reference(s): 14
AB A review with 52 refs. Blood group antigens (BGAs) are chem. moieties on the red blood cell (RBC) membrane. Some BGAs (e.g., A, B, H, Lewis, P, I) are widely distributed throughout the body and may not be primarily erythroid antigens. Statistical correlations with ABO blood groups and disease have been made for years and have been highly controversial. It is not known if BGAs have a biol. function. There are increasing reports of BGAs [e.g., Lex (an isomer of Lea), Ley (an isomer of Leb), T, Tn, "A-like"] appearing as "new" antigens on malignant tissue. Their presence and membrane d. appears to correlate with the metastatic potential of the tumor. This often parallels loss of normal BGAs (e.g., ABH) from the tissue. Some of these antigens have been shown to influence the humoral and cellular response and have been used in assays to det. preclin. cancer, and in tumor immunotherapy. Interactions of some parasites and bacteria with human cells have been shown to depend on the presence of certain BGAs. P. vivax malarial parasites only enter human RBCs when the Fy6 Duffy blood group protein is present on the RBCs. Certain E. coli will only attach to the epithelial cells of the urinary tract if P or Dr BGAs are present in the epithelial cells. The P antigen is also the RBC receptor for Parvovirus B19. Leb has recently been found to be the receptor for **H. pylori** in the gastric tissue. The high frequency BGA, AnWj, is the RBC receptor for H. influenzae. BGAs have been shown to be assocd. closely with some important complement proteins. Ch/Rg BGAs have been found not to be true BGAs but are RBC-bound C4 (C4d). Knops/McCoy/York BGAs have been located on the C3b/C4b receptor (CR1). The high frequency BGAs of the Cromer (Cr) system are located on decay accelerating factor (DAF or CD55). Cartwright (Yt) BGAs are located on RBC acetylcholinesterase mols. DAF and

acetylcholinesterase are on phosphatidylinositol-glycan (PIG) linked proteins. When the PIG anchor is missing from RBCs, as in paroxysmal nocturnal hemoglobinuria, the affected RBCs lack all Cr, Yt, JMH, Hy/Gy, Do and Emm BGAs. The most important ligand for P, E and L selectins is **sialyl-Lex**. This interaction is the tethering stage that start the leukocytes' journey from the circulation into the tissue. It appears that malignant cells may move through tissue in a similar way and may explain the close assocn. of Lex with metastasis. Thus, there are increasing data suggesting a biol. role for BGAs unrelated to the RBC.

ST review blood group antigen tumor disease

IT Bacteria
Neoplasm
Parasite
Virus, animal
(blood group antigens as tumor markers, parasitic/bacterial/viral receptors, and their assocn. with immunol. important proteins)

IT Blood-group substances
RL: ADV (Adverse effect, including toxicity); **BAC (Biological activity or effector, except adverse)**; BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(blood group antigens as tumor markers, parasitic/bacterial/viral receptors, and their assocn. with immunol. important proteins)

IT Receptors
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(blood group antigens as tumor markers, parasitic/bacterial/viral receptors, and their assocn. with immunol. important proteins)

IT Disease
(blood group antigens in relation to disease susceptibility)

L125 ANSWER 21 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1994:626211 HCAPLUS
DN 121:226211
TI Therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro
AU Huesca, M.; Gold, B.; Sherman, P.; Lewin, P.; Lingwood, C.
CS Departments Microbiology, Hospital Sick Children, Toronto, ON, M5G 1X8, Can.
SO Zentralblatt fuer Bakteriologie (**1993**), 280(1-2), 244-52
CODEN: ZEBAE8; ISSN: 0934-8840
DT Journal
LA English
CC 10-5 (Microbial, Algal, and Fungal Biochemistry)
AB Treatment with bismuth-contg. remedies has been long assocd. with the alleviation of minor gastric ailments. Bismuth salts have a potent antimicrobial activity, and are part of the current std. regime used to treat **Helicobacter pylori** infection. **H. pylori** is considered to be the major etiol. factor in the development of peptic ulcer disease. Earlier efficacious treatments for peptic ulcer included the oral administration of Tween detergents. We have found that these agents have an inhibitory effect on **H. pylori** adhesion to the lipid species phosphatidylethanolamine (PE) and gangliotetraosylceramide (Gg4) shown previously to be receptors for **H. pylori** binding in vitro. **H. pylori** binding to PE and Gg4 was inhibited after a thirty minute preincubation with different bismuth compds.: bismuth subsalicylate > bismuth subgallate > bismuth carbonate > colloidal bismuth subcitrate > tripotassium dicitrato bismuthate. No inhibitory effect on **H. pylori** binding was obsd. when bismuth salts were added directly into the binding assay. No changes in bacterial morphol. and motility were obsd. after the thirty minute incubation. Pretreatment with Tween detergents also inhibited **H. pylori** receptor binding by up to 80% at

concns. as low as 0.0001%. These results suggest that inhibition of **H. pylori**/host cell adhesion might play a role in efficacious treatment for this infection.

ST Helicobacter receptor binding inhibition antiulcer agent; bismuth salt inhibition Helicobacter receptor binding; Tween inhibition Helicobacter receptor binding

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Helicobacter pylori**; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Bactericides, Disinfectants, and Antiseptics
(bismuth salts and Tween derivs.; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT **Campylobacter pyloridis**
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Ulcer inhibitors
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Phosphatidylethanolamines
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Adhesion
(bio-, therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT 57644-54-9, Tripotassium dicitrato bismuthate
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(colloidal and noncolloidal; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT 99-26-3, Bismuth subgallate 9005-64-5, Tween 20 9005-65-6, Tween 80 9005-66-7, Tween 40 14882-18-9, Bismuth subsalicylate 16508-95-5, Bismuth carbonate
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

RN 71012-19-6 HCAPLUS

CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 22 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1993:240932 HCAPLUS
DN 118:240932
TI Receptor conjugates for targeting drugs and other agents
IN Krivan, Howard C.; Blomberg, Arne Lennart Ingemar
PA Microcarb Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K047-48
ICS A61K009-127
CC 63-5 (**Pharmaceuticals**)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9302709	A1	19930218	WO 1991-US5422	19910731 <--
	W: CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	EP 598719	A1	19940601	EP 1991-915386	19910731 <--
	EP 598719	B1	19980916		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 06511466	T2	19941222	JP 1991-514489	19910731 <--
	AT 171072	E	19981015	AT 1991-915386	19910731 <--
	ES 2123514	T3	19990116	ES 1991-915386	19910731 <--
	LV 12233	B	19991020	LV 1998-282	19981222 <--
PRAI	WO 1991-US5422	W	19910731 <--		

AB Drugs, esp. anti-infective agents, are coupled to a receptor which binds to a microorganism. The selectivity of the receptor permits increased targeting and specificity for the pathogen. Thus, asialo Gm1-amoxicillin was prepd. and its antibacterial effect was demonstrated with monkeys infected with **Helicobacter pylori**.

ST antibiotic receptor conjugate; asialoganglioside Gm1 amoxicillin conjugate
IT Antibiotics
(conjugates with microorganism receptors, for cell targeting)
IT Receptors
RL: BIOL (Biological study)
(microorganism-binding, anti-infective agent conjugate formation with, for cell targeting)
IT Bacteria
Fungi
Mycoplasma
Parasite
Virus
(receptors of, drug conjugates with, for cell targeting)
IT Steroids, compounds
RL: BIOL (Biological study)
(conjugates, with microorganism receptors, for cell targeting)
IT Pharmaceutical dosage forms
(liposomes, anti-infective agent conjugates with microorganism receptors in)
IT Receptors
RL: BIOL (Biological study)
(pharmaceutical, conjugates with microorganism, for cell targeting)
IT Pharmaceuticals
RL: BIOL (Biological study)
(receptors, conjugates with microorganism, for cell targeting)
IT 26787-78-0, Amoxicillin
RL: PROC (Process)
(conjugate formation of, with asialo Gm2)
IT 26787-78-0DP, reaction products with asialo Gm1 **71012-19-6DP**, reaction products with amoxicillin
RL: **BAC (Biological activity or effector, except adverse)**; BSU

(Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(prepn. and antibacterial activities of)
IT 147686-73-5P
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. and antibacterial activity of)
IT 131070-85-4P 131070-86-5P 131070-87-6P 131070-89-8P 131070-90-1P
131070-92-3P 147662-09-7P 147686-72-4P 147780-81-2P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and reaction of, in prepn. of asialo Gm2)
IT 147662-10-0P 147662-11-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and reaction of, in prepn. of asialo Gm2-amoxicillin conjugate)
IT 463-71-8, Carbonothioic dichloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with asialo Gm2 deriv.)
IT 6291-42-5
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with ethanethiol in prepn. of asialo Gm2)
IT 100-52-7, Benzaldehyde, reactions
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with galactopyranosylthioglucopyranoside in prepn. of asialo Gm2)
IT 108-24-7, Acetic anhydride 407-25-0, Trifluoroacetic anhydride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with glucopyranoside deriv. in prepn. of asialo Gm2)
IT 75-08-1, Ethanethiol
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with lactose peracetate in prepn. of asialo Gm2)
IT 117153-30-7
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with phthalic anhydride in prepn. of asialo Gm2)
IT 85-44-9, 1,3-Isobenzofurandione
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with thiogalactopyranoside deriv. in prepn. of asialo Gm2)
IT 100-27-6, 2-(4-Nitrophenyl)ethanol 104-83-6, p-Chlorobenzyl chloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with thioglucopyranoside deriv. in prepn. of asialo Gm2)
IT **71012-19-6DP**, reaction products with amoxicillin
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(prepn. and antibacterial activities of)
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 23 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1992:658192 HCAPLUS
DN 117:258192
TI Use of host cell phospholipids for inhibiting microbial colonization
IN Krivan, Howard C.; Nilsson, Bo; Lingwood, Clifford A.
PA Microcarb Inc., USA; HSC Research and Development
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2

DT Patent
LA English
IC ICM A61K031-685
ICS A61K031-70
ICA C07H015-10
ICI A61K031-70, A61K031-685
CC 63-3 (**Pharmaceuticals**)
Section cross-reference(s): 9
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9211015	A1	19920709	WO 1991-US9800	19911220	<--
	W: CA, JP					
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE					
	EP 563256	A1	19931006	EP 1992-903046	19911220	<--
	EP 563256	B1	19950628			
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE					
	JP 06511469	T2	19941222	JP 1991-503224	19911220	<--
	JP 3042713	B2	20000522			
	US 5411948	A	19950502	US 1993-78474	19930616	<--
PRAI	US 1990-632372	A	19901221	<--		
	WO 1991-US9800	W	19911220	<--		

AB Inhibition of microbial colonization in a biol. prepn. comprises a phospholipid having the formula: XOCH2CH(OY)CH2OP(O)O-O(CH2)2N+H3 (X = COR, CH:CHR1; Y = COR; R = alkyl, hydroxyalkyl, alkenyl,; R1 = alkyl) in combination with a ceramide deriv. Examples are given on the binding of Chlamydia trachomatis and **Helicobacter pylori** to phospholipids.
ST microbial colonization inhibition phospholipid ceramide deriv
IT Bacteria
Campylobacter pyloridis
Chlamydia trachomatis
Microorganism
(colonization of, in biol. prepns., immobilized host cell phospholipids combination with ceramide derivs. inhibition of)
IT Phospholipids, biological studies
RL: PREP (Preparation)
(immobilized, microbial colonization in biol. prepns. inhibition by ceramide derivs. and)
IT Phosphatidylethanolamines
RL: BIOL (Biological study)
(microbial binding to host cell, as receptor)
IT Brain, composition
Erythrocyte
(phosphatidylethanolamine of, as receptor, microbial binding to)
IT Receptors
RL: BIOL (Biological study)
(phospholipid, of host cells, microbial binding to)
IT **35960-33-9 71012-19-6**
RL: BIOL (Biological study)
(microbial colonization in biol. prepns. inhibition by immobilized host cell phospholipid and)
IT **35960-33-9 71012-19-6**
RL: BIOL (Biological study)
(microbial colonization in biol. prepns. inhibition by immobilized host cell phospholipid and)
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71012-19-6 HCAPLUS

CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 12 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:222395 HCAPLUS
DN 128:321033
TI Inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by bovine colostrum
AU Bitzan, Martin M.; Gold, Benjamin D.; Philpott, Dana J.; Huesca, Mario; Sherman, Philip M.; Karch, Helge; Lissner, Reinhard; Lingwood, Clifford A.; Karmali, Mohamed A.
CS Division of Microbiology, University of Toronto, Ontario, Can.
SO Journal of Infectious Diseases (**1998**), 177(4), 955-961
CODEN: JIDIAQ; ISSN: 0022-1899
PB University of Chicago Press
DT Journal
LA English
CC 18-7 (Animal Nutrition)
Section cross-reference(s): **1**, 15
AB **Helicobacter pylori**, the etiol. agent of chronic-active gastritis and duodenal ulcers in humans, and Helicobacter mustelae, a gastric pathogen in ferrets, bind to phosphatidylethanolamine (PE), a constituent of host gastric mucosal cells, and to gangliotetraosylceramide (Gg4) and gangliotriaosylceramide (Gg3). The effect of a bovine colostrum conc. (BCC) on the interaction of **H. pylori** and H. mustelae to their lipid receptors was examd. BCC blocked attachment of both species to Gg4, Gg3, and PE. Partial inhibition of binding was obsd. with native bovine and human colostra. BCC lacked detectable antibodies (by immunoblotting) to **H. pylori** surface proteins (adhesins). However, colostral lipid exts. contained PE and lyso-PE that bound **H. pylori** in vitro. These results indicate that colostrum can block the binding of Helicobacter species to select lipids and that binding inhibition is conferred, in part, by colostral PE or PE derivs. Colostral lipids may modulate the interaction of **H. pylori** and other adhesin-expressing pathogens with their target tissues.
ST colostrum lipid receptor helicobacter antimicrobial
IT Stomach, disease
(gastritis; inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)
IT Antimicrobial agents
Colostrum
Helicobacter mustelae
Helicobacter pylori
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)
IT Antibodies
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)
IT Adhesins
Phosphatidylethanolamines, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)
IT Receptors

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(lipid; inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT **35960-33-9**, Gangliotriaosylceramide **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

IT **35960-33-9**, Gangliotriaosylceramide **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(inhibition of **Helicobacter pylori** and Helicobacter mustelae binding to lipid receptors by colostrum from humans and cows)

RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 13 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1998:115869 HCAPLUS
DN 128:226251
TI Gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation
IN Murakami, Motoyasu; Hata, Yoshiyuki
PA Murakami, Motoyasu, Japan; Kaken Pharmaceutical Co., Ltd.
SO Jpn. Kokai Tokkyo Koho, 8 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
IC ICM A61K031-70
ICS A61K031-70
CC 1-9 (**Pharmacology**)
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10045602	A2	19980217	JP 1996-202098	19960731 <--

PRAI JP 1996-202098 19960731 <--

AB Gangliosides (GD3, GD1a, GD1b, etc.) are claimed as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation for treatment of stomach diseases including gastritis and ulcer.

ST ganglioside Helicobacter adhesion IL8 antiulcer gastritis

IT Adhesion, biological
Antiulcer agents
Helicobacter pylori
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)

IT Gangliosides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori**

adhesion and interleukin-8 formation)
IT Interleukin 8
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT Stomach, disease
(gastritis; gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT **71012-19-6**, Asialo-Ganglioside GM1 **89678-50-2**, Ganglioside GM3 **98743-26-1** **103220-36-6**, Ganglioside GM1 inner ester **104443-57-4**, Ganglioside GM2 **104443-58-5**, Ganglioside GT1b **104443-59-6**, GD1a **104443-60-9**, GD1b **104443-61-0**, GD3 **104443-62-1**, Ganglioside GM1 **105732-59-0**, Ganglioside GQ1b **107371-09-5**, Ganglioside GD2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
IT **71012-19-6**, Asialo-Ganglioside GM1 **89678-50-2**, Ganglioside GM3 **98743-26-1** **103220-36-6**, Ganglioside GM1 inner ester **104443-57-4**, Ganglioside GM2 **104443-58-5**, Ganglioside GT1b **104443-59-6**, GD1a **104443-60-9**, GD1b **104443-61-0**, GD3 **104443-62-1**, Ganglioside GM1 **105732-59-0**, Ganglioside GQ1b **107371-09-5**, Ganglioside GD2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(gangliosides as inhibitors for **Helicobacter pylori** adhesion and interleukin-8 formation)
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 89678-50-2 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 98743-26-1 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-9-O-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 103220-36-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]-, intramol. 1B1,2B-ester (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-57-4 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-58-5 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-59-6 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-60-9 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-61-0 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 104443-62-1 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 105732-59-0 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-N-acetyl-.alpha.-neuraminosyl-(2.fwdarw.3)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 107371-09-5 HCAPLUS
CN Ceramide, 1-O-[O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.8)-O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-[2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)]-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 14 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1997:745956 HCAPLUS
DN 128:30403
TI Bismuth salts of sialyloligosaccharides and a method for treating and inhibiting gastric and duodenal ulcers using them
IN Swarz, Herbert

PA Neose Technologies, Inc., USA
SO PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-70
ICS A61K031-715; A61K033-24
CC 1-9 (**Pharmacology**)
Section cross-reference(s): **63**

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9741875	A1	19971113	WO 1997-US6376	19970428 <--
	W: AU, CA, JP, KR, MX				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	CA 2253913	AA	19971113	CA 1997-2253913	19970428 <--
	AU 9727326	A1	19971126	AU 1997-27326	19970428 <--
	AU 710576	B2	19990923		
	EP 918526	A1	19990602	EP 1997-921225	19970428 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000509714	T2	20000802	JP 1997-539929	19970428 <--
	KR 2000010732	A	20000225	KR 1998-708842	19981102 <--
PRAI	US 1996-16765P	P	19960503 <--		
	WO 1997-US6376	W	19970428 <--		

AB A method for treating and/or inhibiting gastric and duodenal ulcers comprises administering a pharmaceutical compn. comprising a bismuth salt of an oligosaccharide (NeuAc-.alpha.(2-3)-pGal-.beta.(1)-(X)m-(Y)n-)p-Z, (X = bond or group capable of linking pGal to either linking group Y or multivalent support Z; C1 glycosidic O of galactose may be replaced by N, S, C; Y = linking group; Z = multivalent support; m, n = 0, 1; p = 2-1000) is described. Also described is a method for treating and/or inhibiting gastric and duodenal ulcers, comprising administering a pharmaceutical compn. comprising a bismuth salt of an oligosaccharide NeuAc-.alpha.(2-3)-pGal-.beta.(1)-A (A = group capable of bonding to pGal; C1 glycosidic O of galactose may be replaced by N, S, C).

ST sialyloligosaccharide bismuth salt ulcer inhibitor; gastric ulcer inhibitor sialyloligosaccharide bismuth salt; duodenal ulcer inhibitor sialyloligosaccharide bismuth salt

IT Antihistamines
(H2; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT **Blood-group substances**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**Leb**, **Leb** active oligosaccharide; **sialyloligosaccharide** bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT Dendritic polymers
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Avidins
Lipids, biological studies
Polysaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates, with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

IT Antiulcer agents
(duodenal; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Intestine
(duodenum, **H. pylori** infection; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Drug delivery systems
(enteric; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Stomach, disease
Stomach, disease
(infection, **H. pylori**; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Emulsions
(lipid, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Drug delivery systems
(liposomes, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Drug delivery systems
(oral; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Alcohols, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(polyhydric, conjugates with sialyloligosaccharide bismuth salts; sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Antiulcer agents
Drug delivery systems
Helicobacter pylori
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Fetuins
Sialooligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)
IT Antibacterial agents
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)
IT Antibiotics
Oligosaccharides, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)
IT 12408-02-5, Hydrogen ion, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(proton pump inhibitors; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)
IT 63-42-3, Lactose 7440-69-9D, Bismuth, salts with sialyloligosaccharides, biological studies 9003-05-8D, Polyacrylamide, conjugates with sialyloligosaccharide bismuth salts 9004-54-0D, Dextran, conjugates with sialyloligosaccharide bismuth salts, biological studies 12619-70-4D, Cyclodextrin, conjugates with sialyloligosaccharide bismuth salts

25104-18-1D, Polylysine, conjugates with sialyloligosaccharide bismuth salts **35890-38-1**, 3'-Sialyllactose **35890-38-1D**, 3'-Sialyllactose, albumin conjugates **35890-39-2**, 6'-Sialyllactose 38000-06-5D, Polylysine, conjugates with sialyloligosaccharide bismuth salts 199612-73-2
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

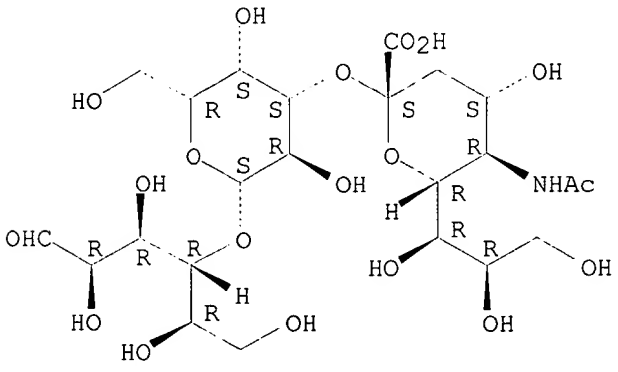
IT 60-54-8D, Tetracycline, derivs. 66357-35-5, Ranitidine 73590-58-6, Omeprazole
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT 12408-02-5, Hydrogen ion, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(transport; inhibitors; sialyloligosaccharide bismuth salts, alone or with other agents, for gastric and duodenal ulcer treatment)

IT **35890-38-1**, 3'-Sialyllactose **35890-38-1D**, 3'-Sialyllactose, albumin conjugates **35890-39-2**, 6'-Sialyllactose
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(sialyloligosaccharide bismuth salts for gastric and duodenal ulcer treatment)

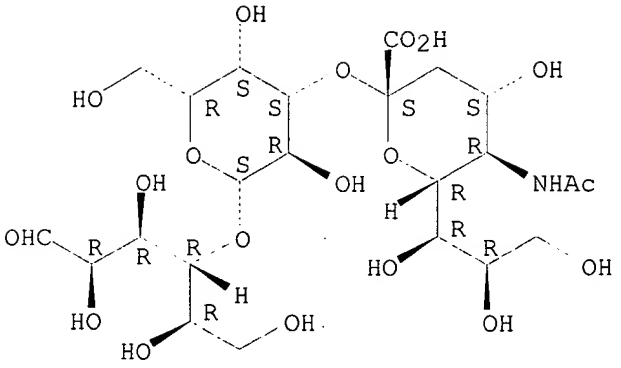
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



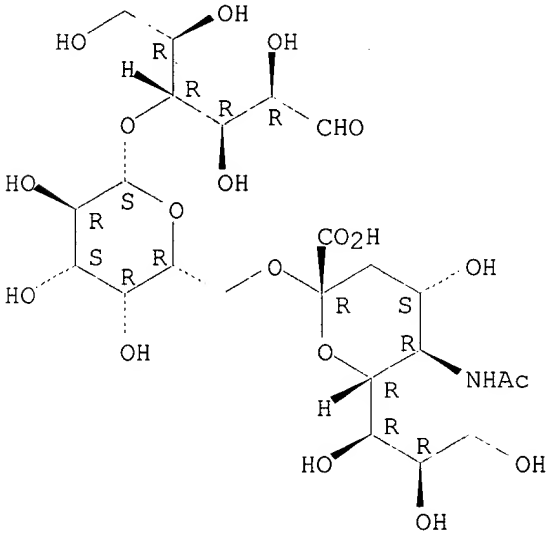
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 35890-39-2 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.6)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L125 ANSWER 16 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:70383 HCAPLUS
DN 124:114313
TI Role of sulfatides in adhesion of **Helicobacter pylori** to gastric cancer cells
AU Kamisago, Satoshi; Iwamori, Masao; Tai, Tadashi; Mitamura, Keiji; Yazaki, Yoshio; Sugano, Kentaro
CS Third Dep. Internal Medicine, Univ. Tokyo, Tokyo, 113, Japan
SO Infection and Immunity (**1996**), 64(2), 624-8
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
CC 14-7 (Mammalian Pathological Biochemistry)
AB We have demonstrated that clin. isolates of **Helicobacter pylori** preferentially bind to sulfatides (I3SO3-GalCer) and GM3 gangliosides (II3NeuAcLacCer), two predominant acidic glycosphingolipids in the human gastric mucosa, on thin-layer chromatog. plates. However, it has not yet been clarified that these glycosphingolipids truly serve as

adhesion receptors for **H. pylori** in live cells. In this study, we used a gastric cancer cell line, KATO III, as a cellular model of **H. pylori** adhesion and examd. the role of sulfatides in attachment. The adhesion of **H. pylori** (i.e., a std. strain of **H. pylori**, NCTC 11637) to KATO III cells and the effects of various substances on this adhesion were monitored and semiquantitated by flow cytometric anal. Sulfated glycoconjugates, such as heparin and gastric mucin, significantly inhibited **H. pylori** adhesion to KATO III cells. Membrane prepns. from KATO III cells strongly inhibited this adhesion. In the membrane prepns., sulfatides were present as a major acidic glycosphingolipid. With the exception of sulfatides, no distinct adhesion of **H. pylori** to glycosphingolipids from KATO III cells was obsd. Moreover, **H. pylori** did not bind to any membrane proteins of KATO III cells. Finally, a monoclonal anti-sulfatide antibody markedly reduced **H. pylori** adhesion to KATO III cells. These results suggest that sulfatides, and possibly related sulfated compds., serve as a major receptor for cell adhesion by **H. pylori**.

ST sulfatide adhesion Helicobacter stomach

IT **Campylobacter pyloridis**
Stomach
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)

IT Sulfatides
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)

IT Mucins
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells inhibition by)

IT Adhesion
(bio-, sulfatides in adhesion of **Helicobacter pylori** to gastric cells)

IT **54827-14-4**, Ganglioside gm3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)

IT 9005-49-6, Heparin, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells inhibition by)

IT **54827-14-4**, Ganglioside gm3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(sulfatides in adhesion of **Helicobacter pylori** to gastric cells)

RN 54827-14-4 HCAPLUS
CN Ganglioside GM3 (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 17 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1996:13184 HCAPLUS
DN 124:76496
TI Asialoganglioside-antibiotic conjugates for treating bacterial infection
IN Krivan, Howard C.; Blomberg, A. Lennart I.
PA MicroCarb, Inc., USA
SO U.S., 12 pp. Cont. of U.S. Ser. No. 484,568, abandoned.
CODEN: USXXAM
DT Patent

LA English
IC ICM A61K031-715
ICS A61K031-705; A61K039-00
NCL 514054000
CC 1-5 (**Pharmacology**)
Section cross-reference(s): 2, 15, **63**
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5466681	A	19951114	US 1994-180397	19940112 <--
PRAI US 1990-484568		19900223 <--		

AB Asialogangliosides, such as asialo-GM1 and asialo-GM2, are used for targeting penicillin antibiotics to bacteria. The present invention provides prepn. of conjugates of the microorganism receptor (i.e. asialo-GM1 and asialo-GM2) and anti-infectives (i.e. antibiotic, steroid, synthetic drugs, or a mol. that can induce prodn. of antibody). The present invention also provides methods for treating infections in warm-blooded animals due to pathogenic microorganisms, e.g. Streptococcus pneumoniae, **Helicobacter pylori**.

ST asialoganglioside antibiotic conjugate bacterial infection

IT Antibiotics
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(conjugates with asialoganglioside; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT Bacteria
Campylobacter pyloridis
Microorganism
Streptococcus pneumoniae
(infection; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT Gangliosides
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(asialo-, conjugates with antibiotics; prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT 131070-85-4P 131070-86-5P 131070-89-8P 131070-90-1P 131070-91-2P
131070-92-3P 131083-69-7P 147662-10-0P 147662-11-1P 147780-81-2P
172723-15-8P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT **71012-19-6DP**, Asialo-GM1, conjugates with amoxicillin
172723-16-9P
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT 1406-05-9D, Penicillin, conjugates with asialoganglioside 26787-78-0D, Amoxicillin, conjugates with asialoganglioside **35960-33-9D**, Asialo-GM2, conjugates with antibiotic
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

IT **71012-19-6DP**, Asialo-GM1, conjugates with amoxicillin
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)

RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
IT **35960-33-9D**, Asialo-GM2, conjugates with antibiotic
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prepn. of asialoganglioside-antibiotic conjugates for treating bacterial infection)
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 18 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:995045 HCAPLUS
DN 124:146728
TI Preparation of synthetic carbohydrate which bind to **Helicobacter pylori** for use as drugs.
IN Danishefsky, Samuel J.; Randolph, John T.
PA Sloan-Kettering Institute for Cancer Research, USA
SO PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM C07H005-02
ICS C07H015-02; C07H015-20; A61K031-715; A61K031-72
CC 33-4 (Carbohydrates)
Section cross-reference(s): **1**
FAN.CNT 7

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9525113	A1	19950921	WO 1995-US3273	19950315 <--
	W: AU, CA, JP, MX				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 5543505	A	19960806	US 1994-213053	19940315 <--
	AU 9521005	A1	19951003	AU 1995-21005	19950315 <--
PRAI	US 1994-213053	A	19940315 <--		
	WO 1995-US3273	W	19950315 <--		

OS MARPAT 124:146728
GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB Title compds. (I; A = amino acid bearing an .omega.-amino group or .omega.-carbonyl group, amino acid residue of a peptide which bears an .omega.-amino group or .omega.-carbonyl group, amino acid residue of a protein which bears an .omega.-amino group or .omega.-carbonyl group; R1 = H, OH, NH2, NHR4; R4 = SO2Ph, alkyl, acyl, aryl; M = Q1; n = 0-18; where n is >1, each M is independently the same or different; p = 0, 1; R2, R3, R5, R6 = H, OH; with the proviso that geminal R2 and R3 are not both OH and geminal R5 and R6 are not both OH; X, Y = H2, O; q .gtoreq.1; with the proviso than when A = amino acid bearing an .omega.-amino group or an .omega.-carbonyl group, q = 1), are claimed for treatment of disorders caused by **Helicobacter pylori** (no data). Thus, conjugatable Lewis Y blood group determinant (II) was prepd. in several steps from lactal (III) via intermediate (IV).
ST oligosaccharide prepn **helicobacter pylori** adhesion inhibitor; ulcer inhibitor oligosaccharide prepn; gastric adenocarcinoma treatment oligosaccharide; blood group determinant conjugatable prepn
IT **Blood-group substances**
RL: **BAC (Biological activity or effector, except adverse)**; BSU

(Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(conjugatable **Lewis** X and Y determinants; prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT **Campylobacter pyloridis**
Neoplasm inhibitors
Ulcer inhibitors
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT Oligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT Stomach, neoplasm
(adenocarcinoma, treatment; prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 173053-82-2P
RL: PNU (Preparation, unclassified); PREP (Preparation)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 98-10-2, Benzenesulfonamide 65207-55-8 127061-08-9 137915-37-8 142800-26-8 145852-76-2 149625-80-9 149847-26-7D, polymer-bound 159494-42-5 173053-78-6 173053-80-0
RL: RCT (Reactant); RACT (Reactant or reagent)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 159494-36-7P 159494-38-9P 162128-74-7P 162128-75-8P 162128-76-9P 162128-80-5P 162128-81-6P 162128-82-7P 162128-84-9P 162128-85-0P 163228-26-0P 163228-28-2P 163228-34-0P 163228-36-2P 173053-77-5DP, polymer-bound 173053-79-7P 173053-81-1P 173053-84-4DP, polymer-bound 173053-85-5DP, polymer-bound 173053-85-5P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

IT 162128-77-0P 163228-29-3P 173053-83-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of synthetic carbohydrates which bind to **Helicobacter pylori** for use as drugs)

L125 ANSWER 19 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1995:893094 HCAPLUS
DN 123:276048
TI Oligosaccharides for treating and inhibiting gastric and duodenal ulcers
IN Zopf, David A.; Simon, Paul M.; Roth, Stephen; Mcguire, Edward J.; Langer, Dennis H.
PA Neose Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-715
CC 1-9 (**Pharmacology**)
Section cross-reference(s): **63**

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9523605	A1	19950908	WO 1995-US2388	19950302 <--

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TT, UA
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2183329	AA	19950908	CA 1995-2183329	19950302	<--
AU 9519323	A1	19950918	AU 1995-19323	19950302	<--
AU 709149	B2	19990819			
EP 749314	A1	19961227	EP 1995-911945	19950302	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE					
JP 09509931	T2	19971007	JP 1995-522955	19950302	<--
JP 3179108	B2	20010625			
US 5514660	A	19960507	US 1995-474199	19950607	<--
US 5753630	A	19980519	US 1996-598431	19960208	<--
US 5883079	A	19990316	US 1998-75862	19980512	<--

PRAI US 1994-204515	A	19940302	<--	
US 1992-922519	B2	19920731	<--	
US 1993-104483	B1	19930728	<--	
WO 1995-US2388	W	19950302	<--	
US 1995-474199	A1	19950607	<--	
US 1996-598431	A1	19960208	<--	

AB A method for treating and/or inhibiting gastric and duodenal ulcers, comprises administering a pharmaceutical compn. comprising an oligosaccharide of the following formula: (NeuAc-.alpha.(2-3)-pGal-.beta.(1)-(-X-)m-(-Y-)n-)p-Z; wherein X is a chem. bond or a group capable of linking the p-galactose to either the linking group Y or the multivalent support Z; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C; Y is a linking group; Z is a multivalent support; m is 0 or 1; n is 0 or 1; and p is an integer of 2-1,000. Also described is a pharmaceutical compn. comprising an oligosaccharide of the formula: NeuAc-.alpha.(2-3)-pGal-.beta.(1)-A wherein A is a group capable of bonding to the p-galactose; wherein the C1 glycosidic oxygen of galactose may be replaced by N, S or C. IC50 value of 3'-sialyl lactose against **Helicobacter pylori** was 6.times.10-3 mmol/mL.
An antiulcer compn. was prepd. by mixing 1g 3'-sialyl lactose and 0.25g ranitidine in water/propylene glycol.

ST ulcer inhibitor oligosaccharide; antiulcer sialyl lactose Helicobacter inhibitor

IT **Campylobacter pyloridis**
(infections; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Ulcer inhibitors
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Fetuins
Oligosaccharides
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Antibiotics
(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Antihistaminics
(H2, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **Blood-group substances**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)

(**Leb**, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Ulcer inhibitors
(duodenal, oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT Pharmaceutical dosage forms
(oral, oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT Albumins, biological studies
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(reaction products, with sialyl lactose; oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

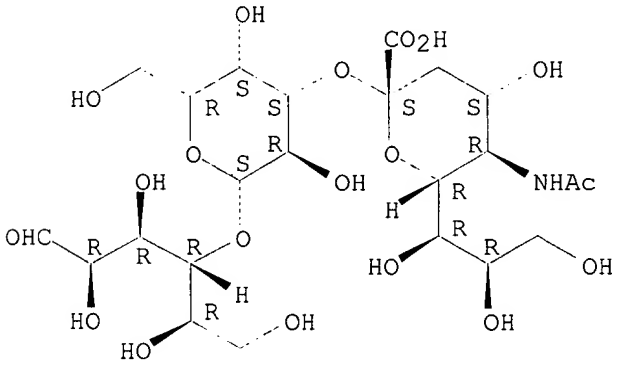
IT **35890-38-1**, 3'-Sialyl lactose **35890-38-1D**, 3'-Sialyl lactose, reaction products with albumins **35890-39-2**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

IT 60-54-8, Tetracycline 66357-35-5, Ranitidine 73590-58-6, Omeprazole
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides in combination with antiulcerative agents for treating and inhibiting gastric and duodenal ulcers)

IT **35890-38-1**, 3'-Sialyl lactose **35890-38-1D**, 3'-Sialyl lactose, reaction products with albumins **35890-39-2**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(oligosaccharides for treating and inhibiting gastric and duodenal ulcers)

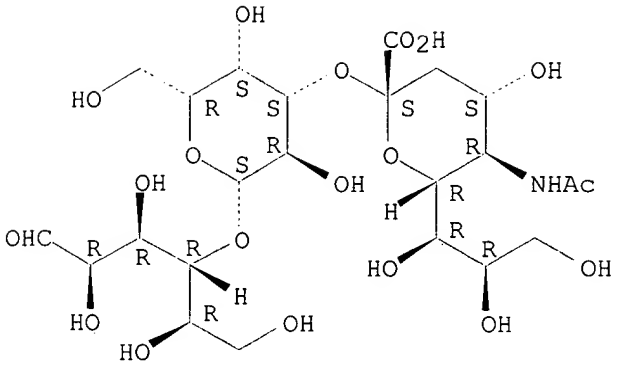
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



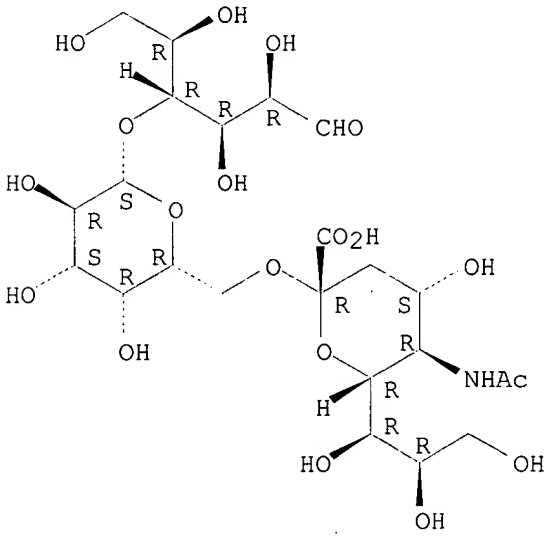
RN 35890-38-1 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.3)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 35890-39-2 HCAPLUS
CN D-Glucose, O-(N-acetyl-.alpha.-neuraminosyl)-(2.fwdarw.6)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L125 ANSWER 21 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1994:626211 HCAPLUS
DN 121:226211
TI Therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro
AU Huesca, M.; Gold, B.; Sherman, P.; Lewin, P.; Lingwood, C.
CS Departments Microbiology, Hospital Sick Children, Toronto, ON, M5G 1X8, Can.
SO Zentralblatt fuer Bakteriologie (**1993**), 280(1-2), 244-52
CODEN: ZEBAE8; ISSN: 0934-8840
DT Journal
LA English
CC 10-5 (Microbial, Algal, and Fungal Biochemistry)
AB Treatment with bismuth-contg. remedies has been long assocd. with the alleviation of minor gastric ailments. Bismuth salts have a potent antimicrobial activity, and are part of the current std. regime used to treat **Helicobacter pylori** infection. **H. pylori** is considered to be the major etiol. factor in the development of peptic ulcer disease. Earlier efficacious treatments for

peptic ulcer included the oral administration of Tween detergents. We have found that these agents have an inhibitory effect on **H. pylori** adhesion to the lipid species phosphatidylethanolamine (PE) and gangliotetraosylceramide (Gg4) shown previously to be receptors for **H. pylori** binding in vitro. **H. pylori** binding to PE and Gg4 was inhibited after a thirty minute preincubation with different bismuth compds.: bismuth subsalicylate > bismuth subgallate > bismuth carbonate > colloidal bismuth subcitrate > tripotassium dicitrato bismuthate. No inhibitory effect on **H. pylori** binding was obsd. when bismuth salts were added directly into the binding assay. No changes in bacterial morphol. and motility were obsd. after the thirty minute incubation. Pretreatment with Tween detergents also inhibited **H. pylori** receptor binding by up to 80% at concns. as low as 0.0001%. These results suggest that inhibition of **H. pylori**/host cell adhesion might play a role in efficacious treatment for this infection.

ST Helicobacter receptor binding inhibition antiulcer agent; bismuth salt inhibition Helicobacter receptor binding; Tween inhibition Helicobacter receptor binding

IT Receptors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**Helicobacter pylori**; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Bactericides, Disinfectants, and Antiseptics
(bismuth salts and Tween derivs.; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT **Campylobacter pyloridis**
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Ulcer inhibitors
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Phosphatidylethanolamines
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT Adhesion
(bio-, therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT 57644-54-9, Tripotassium dicitrato bismuthate
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(colloidal and noncolloidal; therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT 99-26-3, Bismuth subgallate 9005-64-5, Tween 20 9005-65-6, Tween 80 9005-66-7, Tween 40 14882-18-9, Bismuth subsalicylate 16508-95-5, Bismuth carbonate
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)

IT **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H.**

pylori receptor binding in vitro)
IT **71012-19-6**, Gangliotetraosylceramide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(therapeutics used to alleviate peptic ulcers inhibit **H. pylori** receptor binding in vitro)
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 22 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1993:240932 HCAPLUS
DN 118:240932
TI Receptor conjugates for targeting drugs and other agents
IN Krivan, Howard C.; Blomberg, Arne Lennart Ingemar
PA Microcarb Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K047-48
ICS A61K009-127
CC 63-5 (**Pharmaceuticals**)
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9302709	A1	19930218	WO 1991-US5422	19910731 <--
	W: CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE				
	EP 598719	A1	19940601	EP 1991-915386	19910731 <--
	EP 598719	B1	19980916		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 06511466	T2	19941222	JP 1991-514489	19910731 <--
	AT 171072	E	19981015	AT 1991-915386	19910731 <--
	ES 2123514	T3	19990116	ES 1991-915386	19910731 <--
	LV 12233	B	19991020	LV 1998-282	19981222 <--
PRAI	WO 1991-US5422	W	19910731 <--		

AB Drugs, esp. anti-infective agents, are coupled to a receptor which binds to a microorganism. The selectivity of the receptor permits increased targeting and specificity for the pathogen. Thus, asialo Gm1-amoxicillin was prepd. and its antibacterial effect was demonstrated with monkeys infected with **Helicobacter pylori**.
ST antibiotic receptor conjugate; asialoganglioside Gm1 amoxicillin conjugate
IT Antibiotics
(conjugates with microorganism receptors, for cell targeting)
IT Receptors
RL: BIOL (Biological study)
(microorganism-binding, anti-infective agent conjugate formation with, for cell targeting)
IT Bacteria
Fungi
Mycoplasma
Parasite
Virus
(receptors of, drug conjugates with, for cell targeting)
IT Steroids, compounds
RL: BIOL (Biological study)
(conjugates, with microorganism receptors, for cell targeting)
IT Pharmaceutical dosage forms
(liposomes, anti-infective agent conjugates with microorganism

receptors in)
IT Receptors
RL: BIOL (Biological study)
(pharmaceutical, conjugates with microorganism, for cell targeting)
IT Pharmaceuticals
RL: BIOL (Biological study)
(receptors, conjugates with microorganism, for cell targeting)
IT 26787-78-0, Amoxicillin
RL: PROC (Process)
(conjugate formation of, with asialo Gm2)
IT 26787-78-0DP, reaction products with asialo Gm1 **71012-19-6DP**, reaction products with amoxicillin
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(prepn. and antibacterial activities of)
IT 147686-73-5P
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. and antibacterial activity of)
IT 131070-85-4P 131070-86-5P 131070-87-6P 131070-89-8P 131070-90-1P 131070-92-3P 147662-09-7P 147686-72-4P 147780-81-2P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and reaction of, in prepn. of asialo Gm2)
IT 147662-10-0P 147662-11-1P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and reaction of, in prepn. of asialo Gm2-amoxicillin conjugate)
IT 463-71-8, Carbonothioic dichloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with asialo Gm2 deriv.)
IT 6291-42-5
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with ethanethiol in prepn. of asialo Gm2)
IT 100-52-7, Benzaldehyde, reactions
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with galactopyranosylthioglucopyranoside in prepn. of asialo Gm2)
IT 108-24-7, Acetic anhydride 407-25-0, Trifluoroacetic anhydride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with glucopyranoside deriv. in prepn. of asialo Gm2)
IT 75-08-1, Ethanethiol
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with lactose peracetate in prepn. of asialo Gm2)
IT 117153-30-7
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with phthalic anhydride in prepn. of asialo Gm2)
IT 85-44-9, 1,3-Isobenzofurandione
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with thiogalactopyranoside deriv. in prepn. of asialo Gm2)
IT 100-27-6, 2-(4-Nitrophenyl)ethanol 104-83-6, p-Chlorobenzyl chloride
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction of, with thioglucopyranoside deriv. in prepn. of asialo Gm2)
IT **71012-19-6DP**, reaction products with amoxicillin
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); BIOL (Biological study); PREP (Preparation)
(prepn. and antibacterial activities of)
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-

2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

L125 ANSWER 23 OF 23 HCAPLUS COPYRIGHT 2003 ACS
AN 1992:658192 HCAPLUS
DN 117:258192
TI Use of host cell phospholipids for inhibiting microbial colonization
IN Krivan, Howard C.; Nilsson, Bo; Lingwood, Clifford A.
PA Microcarb Inc., USA; HSC Research and Development
SO PCT Int. Appl., 31 pp.
CODEN: PIXXD2
DT Patent
LA English
IC ICM A61K031-685
ICS A61K031-70
ICA C07H015-10
ICI A61K031-70, A61K031-685
CC 63-3 (**Pharmaceuticals**)
Section cross-reference(s): 9
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9211015	A1	19920709	WO 1991-US9800	19911220	<--
	W: CA, JP					
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, MC, NL, SE					
	EP 563256	A1	19931006	EP 1992-903046	19911220	<--
	EP 563256	B1	19950628			
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, MC, NL, SE					
	JP 06511469	T2	19941222	JP 1991-503224	19911220	<--
	JP 3042713	B2	20000522			
	US 5411948	A	19950502	US 1993-78474	19930616	<--
PRAI	US 1990-632372	A	19901221	<--		
	WO 1991-US9800	W	19911220	<--		

AB Inhibition of microbial colonization in a biol. prepn. comprises a phospholipid having the formula: XOCH2CH(OY)CH2OP(O)O-O(CH2)2N+H3 (X = COR, CH:CHR1; Y = COR; R = alkyl, hydroxyalkyl, alkenyl,; R1 = alkyl) in combination with a ceramide deriv. Examples are given on the binding of Chlamydia trachomatis and **Helicobacter pylori** to phospholipids.
ST microbial colonization inhibition phospholipid ceramide deriv
IT Bacteria
Campylobacter pyloridis
Chlamydia trachomatis
Microorganism
(colonization of, in biol. prepns., immobilized host cell phospholipids combination with ceramide derivs. inhibition of)
IT Phospholipids, biological studies
RL: PREP (Preparation)
(immobilized, microbial colonization in biol. prepns. inhibition by ceramide derivs. and)
IT Phosphatidylethanolamines
RL: BIOL (Biological study)
(microbial binding to host cell, as receptor)
IT Brain, composition
Erythrocyte
(phosphatidylethanolamine of, as receptor, microbial binding to)
IT Receptors
RL: BIOL (Biological study)
(phospholipid, of host cells, microbial binding to)
IT **35960-33-9 71012-19-6**
RL: BIOL (Biological study)

(microbial colonization in biol. prepns. inhibition by immobilized host cell phospholipid and)
IT **35960-33-9 71012-19-6**
RL: BIOL (Biological study)
(microbial colonization in biol. prepns. inhibition by immobilized host cell phospholipid and)
RN 35960-33-9 HCAPLUS
CN Ceramide, 1-O-[O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
RN 71012-19-6 HCAPLUS
CN Ceramide, 1-O-[O-.beta.-D-galactopyranosyl-(1.fwdarw.3)-O-2-(acetylamino)-2-deoxy-.beta.-D-galactopyranosyl-(1.fwdarw.4)-O-.beta.-D-galactopyranosyl-(1.fwdarw.4)-.beta.-D-glucopyranosyl]- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

=> d his

(FILE 'HOME' ENTERED AT 08:49:44 ON 12 MAR 2003)
SET COST OFF

FILE 'REGISTRY' ENTERED AT 08:50:01 ON 12 MAR 2003
L1 2 S 92448-22-1 OR 98603-84-0
L2 0 S (92448-22-1 OR 98603-84-0)/CRN

FILE 'HCAPLUS' ENTERED AT 08:59:17 ON 12 MAR 2003
L3 374 S L1
L4 1450 S SLEX OR SLEA OR SLEWX OR SLEWA OR (SLEW OR SLEWIS)()(X OR A)
L5 9 S SA()(LEX OR LEA OR (LEW OR LEWIS)()(X OR A))
L6 5 S SIAL? ACID()(LEX OR LEA OR (LEW OR LEWIS)()(X OR A))
L7 474 S SIAL?()(LEWISX OR LEWISA)
L8 3 S SIALYLEX OR SIALYLEA OR SIALYLLEWISX OR SIALYLLEWISA OR SIALY
L9 1707 S L3-L8
E TENEBERG S/AU
L10 57 S E3,E4
E HAMMARSTROM L/AU
L11 106 S E3-E8,E17,E18
E HAMMARSTROEM L/AU
L12 93 S E3-E5,E14
E KARLSSON K/AU
L13 325 S E3,E4,E17-E20
E BOREN T/AU
L14 36 S E3-E5
E BOEREN T/AU
L15 8 S L9 AND L10-L14
E WO2000-SE514/AP,RPN
L16 1 S E3
E SE99-1007/AP,PRN
L17 1 S E4
L18 1 S L16,L17 AND L3-L15
SEL RN

FILE 'REGISTRY' ENTERED AT 09:07:00 ON 12 MAR 2003
L19 27 S E1-E27
L20 9 S L19 AND OC5/ES
L21 18 S L19 NOT L20
L22 10 S L21 AND CERAMIDE
L23 19 S L20,L22
L24 1 S 32181-59-2

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     FILE 'HCAPLUS' ENTERED AT 09:10:52 ON 12 MAR 2003
L25         696 S L24
L26        1327 S N()(ACETYLLACTOSAMINE OR ACETYL LACTOSAMINE)
L27          16 S L10-L15 AND L25,L26
L28          23 S L15-L18,L27
L29          22 S L28 NOT L18
                SEL RN

     FILE 'REGISTRY' ENTERED AT 09:13:35 ON 12 MAR 2003
L30         175 S E28-E202
L31         165 S L30 NOT L19
L32         164 S L31 NOT L1
L33          70 S L32 AND OC5/ES
L34          86 S L32 AND UNSPECIFIED
L35          75 S L34 NOT SQL/FA
L36          66 S L35 AND CERAMIDE
L37           9 S L35 NOT L36
L38          76 S L22,L36
                E CERAMIDE
L39        1565 S E3
L40        1375 S L39 NOT SQL/FA
L41        1346 S L40 AND UNSPECIFIED
L42          29 S L40 NOT L41
L43           4 S L42 AND OC5/ES
L44          79 S L41 NOT MAN/CI
L45          73 S L44 NOT (MXS/CI OR COMPD OR WITH)
L46           6 S L44 NOT L45
L47        1263 S L41 AND 1/NC
L48          83 S L41 NOT L47
L49           4 S L48 NOT L42-L46
L50          20 S L34 NOT L36
L51          18 S L23 NOT L1,L24

     FILE 'HCAPLUS' ENTERED AT 09:27:19 ON 12 MAR 2003

     FILE 'REGISTRY' ENTERED AT 09:27:28 ON 12 MAR 2003

     FILE 'HCAPLUS' ENTERED AT 09:32:20 ON 12 MAR 2003
                E BLOOD-GROUP SUBSTANCES/CT
L52        1644 S E17-E23
                E E3+ALL
L53        1738 S E3(L)(LE OR LEA OR LEX OR LEW? OR SIAL?)
L54         279 S E3 (L) FUCOS?
L55          22 S L10-L15 AND L52-L54
L56        4477 S L9,L25,L26,L52-L54
                E HELICOP/CT
                E HELICOB/CT
L57        5084 S E28-E29
                E E28+ALL
L58        6293 S E6,E5+NT
L59        7533 S E5/BI OR E6/BI OR E7/BI OR E8/BI
L60        7666 S (H OR C OR HELICOBACT? OR CAMPYLOBACT?)()PYLORI?
L61         116 S L56 AND L57-L60
                E ADHESINS/CT
                E E3+ALL
L62          27 S L56 AND E4,E5,E3+NT
                E E10+ALL
L63         180 S L56 AND E2+NT
L64         261 S L56 AND E1+NT
                E EPITHELIUM/CT
                E E20+ALL
L65         925 S E2
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                E EPITHELIUM/CT
                E E22+ALL
L66          146 S E2
                E EPITHELIUM/CT
                E E30+ALL
L67         5644 S E2
L68          209 S E4
                E EPITHELIUM/CT
                E E53+ALL
L69         1158 S E2
                E EPITHELIUM/CT
                E E59+ALL
L70           53 S E2

     FILE 'REGISTRY' ENTERED AT 09:44:26 ON 12 MAR 2003
                E EPITHELIUM SMALL INTESTINE/CN

     FILE 'HCAPLUS' ENTERED AT 09:44:26 ON 12 MAR 2003
                E EPITHELIUM SMALL INTESTINE/CT
                E E3+ALL
L71          659 S E2
                E EPITHELIUM SMALL INTESTINE/CT
                E GASTRIC MUCOSA/CT
                E E3+ALL
L72         7298 S E2
L73          101 S E10
L74           67 S L56 AND L65-L73
                E DIGESTIVE TRACT/CT
                E E3+ALL
L75          741 S E3+NT AND L56
                E DIGESTIVE TRACT/CT
                E ULCER/CT
L76         2089 S E5,E7,E8,E10
L77          290 S E15,E16,E17,E18
                E E3+ALL
L78         9575 S E3,E2
                E E4+ALL
L79         5828 S E4,E3,E8-E11
L80          749 S L56 AND L75-L79
L81           62 S L61 AND L62-L64,L74,L80
L82           14 S L81 AND ?FUCO?
L83           39 S L61 AND ?FUCO?
L84           39 S L82,L83
L85           30 S L84 AND (PD<=20000316 OR PRD<=20000316 OR AD<=20000316)
L86            9 S L84 NOT L85
L87           23 S L28,L29
L88           40 S L55,L87
L89           40 S L88 AND L56
L90           23 S L89 AND L57-L84
L91           17 S L89 NOT L90
L92           46 S L85,L90
L93           40 S L92 AND (PD<=20000316 OR PRD<=20000316 OR AD<=20000316)
L94           23 S L92 AND L10-L14
L95           23 S L93 NOT L94
L96          357 S L25,L26 (L) FUCO?
L97           14 S L96 AND L57-L60
L98            2 S L96 AND PHARMACEUT?/SC,SX
L99           16 S L96 AND PHARMACOL?/SC,SX
L100          17 S L98,L99
L101          24 S L25,L26 (L) THU/RL
L102          23 S L101 NOT L97-L100
                SEL DN AN 1 4
L103           2 S E1-E6
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L104          23 S L94,L103

     FILE 'REGISTRY' ENTERED AT 10:08:42 ON 12 MAR 2003

     FILE 'HCAPLUS' ENTERED AT 10:09:00 ON 12 MAR 2003
L105           2 S WO9500527/PN OR WO9523605/PN
L106           1 S VIRCHOWS?/JT AND 1998/PY AND (433 AND 419)/SO
L107           1 S  SCIENCE?/JT AND 1993/PY AND (262 AND 1892)/SO
L108           3 S L105-L107 NOT L104
L109           3 S L108 AND L3-L18,L25-L29,L52-L104

     FILE 'WPIX' ENTERED AT 10:15:28 ON 12 MAR 2003
                 E WO2000-SE514/AP,PRN
L110           1 S E3

     FILE 'HCAPLUS' ENTERED AT 10:22:25 ON 12 MAR 2003

     FILE 'REGISTRY' ENTERED AT 10:23:22 ON 12 MAR 2003
L111        1344 S L45 OR L47 OR L51

     FILE 'HCAPLUS' ENTERED AT 10:25:19 ON 12 MAR 2003
L112       10880 S L111
L113          61 S L112 AND L57-L60
L114         156 S L61 OR L113
L115         110 S L114 AND (PD<=20000316 OR PRD<=20000316 OR AD<=20000316)
L116          99 S L115 NOT L104
L117          24 S L116 AND ?FUCO?
L118          99 S L114 AND L9,L52-L54
L119          27 S L25,L26 AND L57-L60
L120         156 S L114,L119
L121         156 S L114 OR L120
L122          75 S L116 NOT L117
L123          14 S L122 AND (PHARMACEUT? OR PHARMACOL?)/SC,SX
L124          20 S L122 AND (THU OR BAC OR PAC OR PKT)/RL
L125          23 S L123,L124

=> s l120 not l104,l125,l117
L126          92 L120 NOT (L104 OR L125 OR L117)

=> sav l126 fonda937110/a
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